CLINICAL PROFILE OF PERICARDIAL DISEASES

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I hereby declare that the thesis entitled "**CLINICAL PROFILE OF PERICARDIAL DISEASES**" submitted towards partial fulfillment of the requirements for the award of **MD in Medicine** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr Sandeep Chhabra**, Associate Professor, Dayanand Medical College & Hospital, Ludhiana. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family

&

Teachers

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ABBREVIATIONS

S.No.	List of Abbrevations
1	ADA-adenosine deaminase
2	AIDS-Acquired immunodeficiency syndrome
3	AML-acute myeloid leukemia
4	ATT- Anti-tubercular therapy
5	CA-carcinoma
6	CKD-Chronic Kidney Disease
7	CP-Constrictive pericarditis
8	CRP-C-reactive protein
9	cTnI- cardiac troponin I
10	ECHO-echocardiography
11	ESRD- End stage renal disease
12	ESR-erythrocyte sedimentation rate
13	GIST-Gastro-intestinal stromal tumour
14	HFpEF-Heart failure with preserved ejection fraction
15	HFrEF- Heart failure with reduced ejection fraction
16	HIV-human immunodeficiency virus
17	HsCRP-high sensitivity C reactive protein
18	IHD- Ischemic heart disease
19	LVEF-left ventricular ejection fraction
20	MI- myocardial infarction
21	MRSA- Methicillin resistant *Staphylococcus aureus*
22	NHL-Non Hodgkin Lymphoma
23	PD-Pericardial diseases
24	RHD-Rheumatic heart disease
25	RRT- Renal replacement therapy
26	SCC-squamous cell carcinoma
27	SLE-Sytemic lupus erythematosus
28	SOB-shortness of breath
29	TB- Tuberculosis

ABSTRACT

Introduction: Pericardial diseases represent a wide spectrum of clinic-pathological entities such as acute pericarditis, pericardial effusion, cardiac tamponade, and constrictive pericarditis.

Aim of Study:

- To study the clinical spectrum of pericardial diseases.
- To study etiopathogenesis, clinical and echocardiographic profile of patients with pericardial disease.

Materials & Methods: This is an observational study and duration of study was from January 2020 to June 2021. All patients admitted in Medicine/Cardiology department of Dayanand Medical College & Hospital, Ludhiana with diagnosis of pericardial diseases were included in the study.

Results: A total of 170 patients with pericardial disease were included in this study. There were 100 males (58.8%) and 70 females (41.2%) and the mean age of presentation was 54.7 ± 16.3 years. Pericardial effusion was the most common manifestation i.e. in 71.8% followed by acute pericarditis, effusive-pericarditis and constrictive pericarditis in 19.8%, 7.1 % and 1.8% respectively. Shortness of breath, chest pain and fever were the most common presenting symptoms. The most common etiology of pericardial diseases came out to be tubercular. Cardiac tamponade was found in 11.8% (n=20) patients.

Conclusion: Our study demonstrated tuberculosis as the most frequent etiology of pericardial diseases and also the leading cause of tamponade. High prevalence of tuberculosis in India is attributable to poor socio-economic status and overcrowding.

Keywords: Pericarditis, Tamponade, Pericardiocentesis, Constrictive, Effusion.

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INTRODUCTION

The pericardium is a relatively avascular fibrous sac that surrounds the heart. It consists of 2 layers: the visceral and parietal pericardium. The visceral pericardium is composed of a single layer of mesothelial cells that are adherent to the cardiac epicardium. The parietal pericardium is a fibrous structure that is ≤ 2 mm thick and is composed primarily of collagen and a lesser amount of elastin. The 2 layers of the pericardium are separated by a potential space that can normally contain 15 to 35 mL of serous fluid distributed mostly over the atrial-ventricular and interventricular grooves.

The pericardium surrounds the heart and attaches to the sternum, the diaphragm, and the anterior mediastinum and is invested around the great vessels and the venae cavae, serving to anchor the heart in the central thorax. Because of its location, the pericardium may also function as a barrier to infection. The pericardium is well innervated such that the pericardial inflammation may produce severe pain and trigger vagally mediated reflexes. The pericardium also secretes prostaglandins that modulate cardiac reflexes and coronary tone. As a result of its relatively inelastic physical properties, the pericardium limits acute cardiac dilatation and enhances mechanical interactions of the cardiac chambers [1].

Diseases of the pericardium can present clinically in one of several ways:

- Acute and recurrent pericarditis
- Pericardial effusion without major hemodynamic compromise
- Cardiac tamponade
- Constrictive pericarditis
- Effusive-constrictive pericarditis

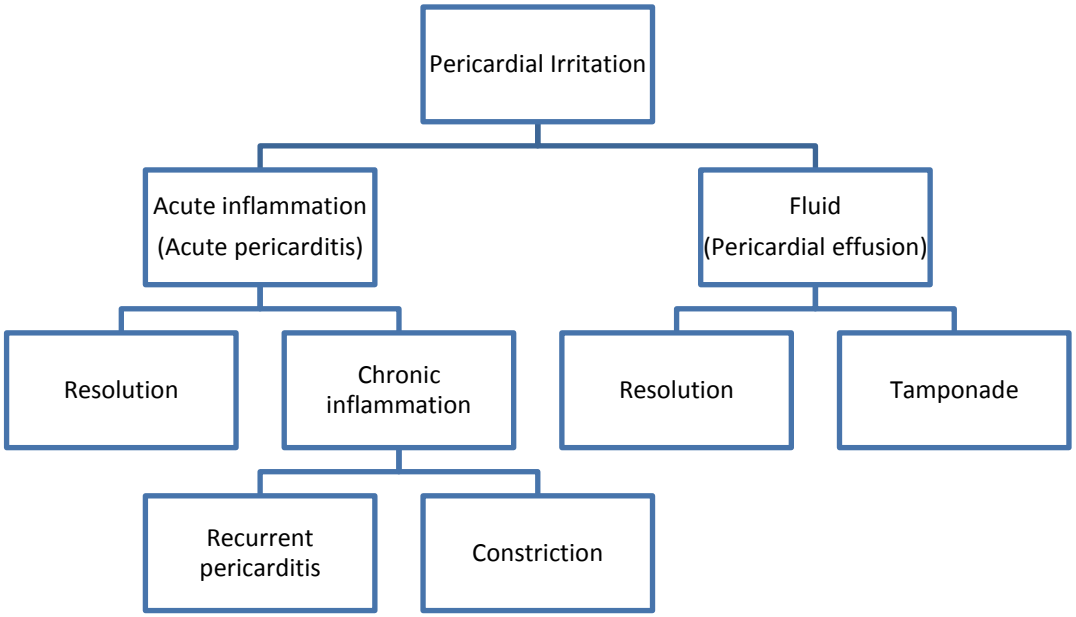

Fig.1: Stage of pericardial disease progression.[2]

ACUTE PERICARDITIS

Acute inflammation of the pericardium with or without an associated pericardial effusion can occur as an isolated clinical problem or as a manifestation of systemic diseases. Although as many as 90% of isolated cases of acute pericarditis are idiopathic or viral, the list of other potential causes is extensive (Table 1). Although formerly common, tuberculous and bacterial infections have become rare causes of pericarditis. Other causes of acute pericarditis include uremia, collagen vascular diseases, neoplasms, and pericardial inflammation after an acute myocardial infarction or pericardial injury. Pericarditis after an acute myocardial infarction most commonly occurs 1 to 3 days after transmural myocardial infarction presumably because of the interaction of the healing necrotic epicardium with the overlying pericardium. A second form of pericarditis associated with myocardial infarction (Dressler's syndrome) typically occurs weeks to months after a myocardial infarction. It is similar to the pericarditis that may occur days to months after traumatic pericardial injury, after surgical manipulation of the pericardium, or after a pulmonary infarction. This syndrome is presumed to be mediated by an autoimmune mechanism and is associated with signs of systemic inflammation, including fever,

and polyserositis. The frequency of pericarditis after myocardial infarction has been reduced by the use of reperfusion therapy.[1]

Idiopathic
Infections (viral, tuberculosis, fungal)
Uremia
Acute myocardial infarction (acute, delayed)
Neoplasm
Post– cardiac injury syndrome (trauma, cardiothoracic surgery)
Systemic autoimmune disease (systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, systemic sclerosing periarteritis nodosa, Reiter's syndrome)
After mediastinal radiation

Table 1: Causes of Acute Pericarditis.[1]

CHRONIC PERICARDITIS

Chronic (>3months) pericarditis includes effusive(inflammatory or hydropericardium in heart failure), adhesive, and constrictive forms. Symptoms are usually mild (chest pain, palpitations, fatigue) and are related to the degree of cardiac compression and pericardial inflammation.[3]

RECURRENT PERICARDITIS

The term recurrent encompasses (1) the intermittent type (symptom free intervals without therapy) and (2) the incessant type (discontinuation of anti-inflammatory therapy ensures a relapse). Massive pericardial effusion, overt tamponade or constriction are rare. Evidence for an immunopathological process include: (1) the latent period lasting for months; (2) the presence of anti-heart antibodies; (3) the quick response to steroid treatment and the similarity and co-existence of recurrent pericarditis with other autoimmune conditions.[3,4]

PERICARDIAL EFFUSION

Pericardial effusion is a common finding in clinical practice either as incidental finding or manifestation of a systemic or cardiac disease. The spectrum of pericardial effusions ranges from mild asymptomatic

effusions to cardiac tamponade. Moreover, pericardial effusion may accumulate slowly or suddenly. Loculated effusions are more common when scarring has supervened (e.g. postsurgical, posttrauma, purulent pericarditis). Massive chronic pericardial effusions are rare(2-3.5% of all large effusions).

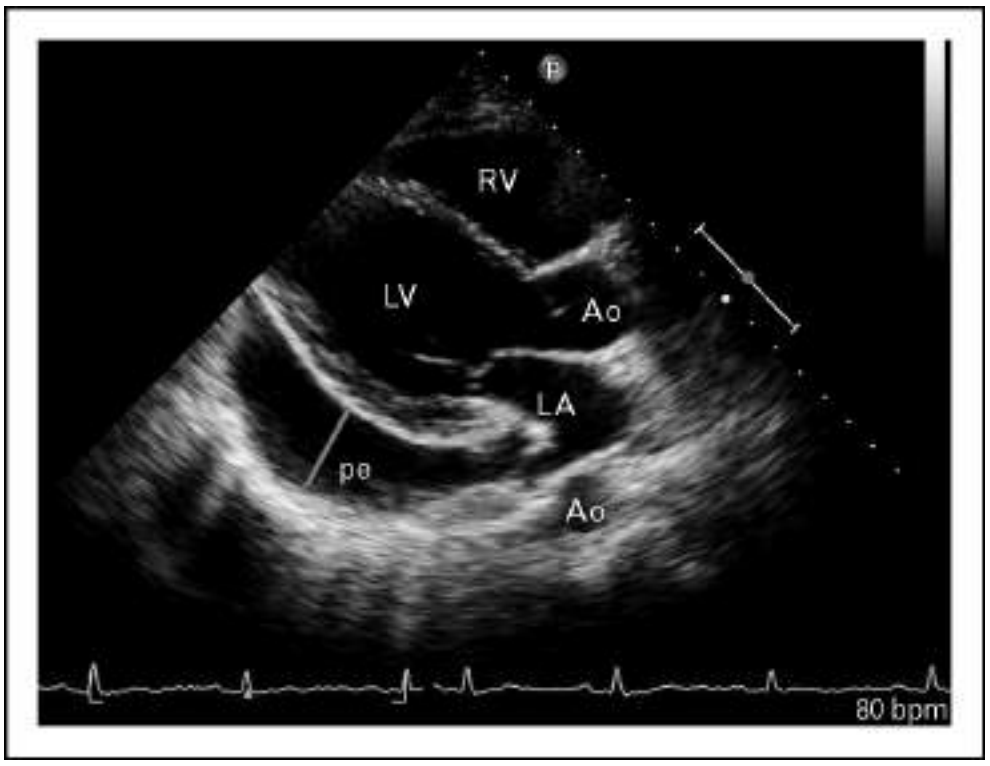

Fig. 2: A simple semiquantitative assessment of pericardial effusion (pe) that may be adopted for either circumferential or loculated effusion is to consider the largest size of pericardial effusion in several views, also considering off-axis views, especially for loculated effusion. The effusion is considered small under 10 mm, moderate from 10 to 20 mm, and large with a diameter more than 20 mm. Ao, aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.[5]

CARDIAC TAMPONADE

Cardiac tamponade is defined as the decompensated phase of cardiac compression caused by a hemodynamically significant effusion. The effusion may be serous, purulent, sanguinous, chylous or pneumatic. Pericardial disease of almost any etiology can produce cardiac tamponade. It is more common in patients with as specific etiology, such as neoplastic, tuberculous or purulent pericarditis. The primary abnormality is compression of all cardiac chambers due to fluid accumulation in the intrapericardial space, resulting in increased intrapericardial pressure sufficient to raise the pressure surrounding the heart to the point where cardiac filling is altered. The right side of the

heart is most vulnerable to compression by a pericardial effusion because of its lower pressures, and abnormal right heart filling is the earliest sign of a hemodynamically significant pericardial effusion.[2]

CONSTRICTIVE PERICARDITIS

Pericardial constriction occurs when a scarred, thickened, and frequently calcified pericardium impairs cardiac filling, limiting the total cardiac volume. The pathophysiological hallmark of pericardial constriction is equalization of the end-diastolic pressures in all 4 cardiac chambers. This occurs because the filling is determined by the limited pericardial volume, not the compliance of the chambers themselves. Initial ventricular filling occurs rapidly in early diastole as blood moves from the atria to the ventricles without much change in the total cardiac volume. However, once the pericardial constraining volume is reached, diastolic filling stops abruptly. This results in the characteristic dip and plateau of ventricular diastolic pressures. The stiff pericardium also isolates the cardiac chambers from respiratory changes in intrathoracic pressures, resulting in Kussmaul's sign.[1] The most frequent causes are mediastinal radiation, chronic idiopathic pericarditis, post cardiac surgery, and tuberculous pericarditis.

EFFUSIVE CONSTRICTIVE PERICARDITIS

Hancock[6] was the first to notice that after removing the pericardial fluid, some patients with cardiac tamponade still had an elevated right atrial pressure. Pericardiocentesis changed the hemodynamics of these patients from those of tamponade to those of constriction. As a result, the restriction of ventricular filling was caused not only by pericardial effusion but also by pericardial constriction (predominantly the visceral pericardium). Idiopathic pericarditis, as well as malignancies and radiation, were the most common causes. Effusive constrictive pericarditis is most likely a stage between acute pericarditis with pericardial effusion and pericardial constriction.[1]

AIMS & OBJECTIVES

- To study the clinical spectrum of pericardial diseases.
- To study etiopathogenesis, clinical and echocardiographic profile of patients with pericardial disease

REVIEW OF LITERATURE

Pericardial disease is a common disorder seen in varying clinical settings and may be the first manifestation of an underlying systemic disease. It may be due to multiple causes. Epidemiologic studies are lacking, and the exact incidence and prevalence are unknown. New diagnostic techniques have improved diagnosis, allowing early diagnosis and management. There are few randomized data to guide physicians in the management of pericardial diseases.[2]

EPIDEMIOLOGY

Despite the relatively high prevalence of pericardial illnesses, epidemiological data, particularly from primary care, is scarce. Pericarditis is by far the most frequent pericardial condition seen in clinical practise. Pericarditis accounts for 0.1 percent of all hospital admissions and 5% of all emergency room visits for chest discomfort. According to Finnish national registry data (2000–9), the standardized incidence rate of hospitalizations for acute pericarditis is 3.32 per 100,000 person-years. These data were limited to hospitalized patients and therefore may account for only a minority of cases, as many patients with pericarditis are commonly not admitted to hospital. Men ages 16–65 years were at higher risk for pericarditis (relative risk 2.02) than women in the general admitted population, with the highest risk difference among young adults compared with the overall population. Acute pericarditis caused 0.20% of all cardiovascular admissions. The in-hospital mortality rate for acute pericarditis was 1.1% and was increased with age and severe co-infections (pneumonia or septicaemia). However, this is a study based on hospital admissions only. Recurrences affect about 30% of patients within 18 months after a first episode of acute pericarditis.[3]

ETIOLOGY

A simple aetiological classification for pericardial diseases can be divided into infectious and non-infectious causes. The aetiology is varied and depends on the epidemiological background, patient

population and clinical setting. In developed countries, viruses are usually the most common aetiological agents of pericarditis, whereas tuberculosis (TB) is the most frequent cause of pericardial diseases in the rest of the world and in developing countries, where TB is endemic. In this setting, TB is often associated with human immunodeficiency virus (HIV) infection, especially in sub-Saharan Africa.[3]

In immunologically competent patient with classic acute pericarditis, the probability of acute idiopathic pericarditis is approximately 90%. Serological data may not prove infection and symptoms usually subside in a few days; in such cases, no further investigations are needed. Specific pericardial investigations may be necessary in a few patients with acute pericardial disease who present with hemodynamic compromise or a protracted clinical course.[2]

Systemic lupus erythematosus (SLE) is probably the best known systemic inflammatory disease. All components of the heart can be affected. Symptomatic pericarditis occurs in about 25% of the patients and asymptomatic pericardial involvement in more than half of the cases. Older autopsy studies reported pericardial involvement in up to 80% of the patients. Tamponade occurs in less than 2% and constrictive pericarditis is extremely rare.[7]

Pericardial involvement in end-stage renal disease (ESRD) is manifested most commonly as acute uremic or dialysis pericarditis and infrequently as chronic constrictive pericarditis. The causes of uremic and dialysis pericarditis remain uncertain. The clinical and laboratory manifestations of acute pericarditis, pericardial effusion, cardiac tamponade, and constrictive pericarditis in patients with chronic renal failure are similar to those observed in nonuremic patients with similar pericardial involvement, except that chest pain occurs less frequently in those with ESRD.[8,9]

The cause of acute pericarditis is either viral or unknown in 9 out of 10 cases (idiopathic). Pericarditis develops in the remaining patients

after a transmural myocardial infarction, in conjunction with other infectious agents, in conjunction with a dissecting aortic aneurysm (in which blood leaks into the pericardial space), after blunt or sharp chest trauma, neoplastic invasion of the pericardium, after chest irradiation, in association with uremia, after cardiac or other thoracic surgery.[10]

Zayas R et al have done a study to access the specific etiology and the role of methods for specific etiologic diagnosis in patients with primary acute pericarditis. Study was done on 100 patients with primary acute pericarditis in which general protocol was performed in all patients but only pericardiocentesis was performed in patients with clinical cardiac tamponade or an unfavourable course with anti-inflammatory drugs. Surgical drainage and pericardial biopsy was performed in patients with tamponade relapse. A specific etiology was discovered in 22 patients (22%) (neoplasm in 7, tuberculosis in 4, other infections in 3, collagen diseases in 3, thyroid disorder in 4 and dissecting aortic aneurysm in 1). Conclusion of this study was that the specific etiology in patients with primary acute pericarditis is about 20% to 25%, and that about 90% of these specific cases can be discovered by using the described systemic diagnostic protocol only in patients with an unfavourable outcome(cardiac tamponade or poor clinical outcome).[11]

Pankuweit S et al concluded that bacterial pericarditis occurs by direct infection during trauma, thoracic surgery, or catheter drainage, by spread from an intrathoracic, myocardial, or subdiaphragmatic focus, and by hematogenous dissemination. The frequent causes are *Staphylococcus* and *Streptococcus* (rheumatic pancarditis), *Haemophilus*, and *M. tuberculosis*. In AIDS pericarditis, the incidence of bacterial infection is much higher than in the general population, with a high proportion of *Mycobacterium avium-intracellulare* infection. Tuberculous infection may present as acute pericarditis, cardiac tamponade, silent (often large) relapsing pericardial effusion, effusive constrictive pericarditis, toxic symptoms with persistent fever, and

acute, subacute, or chronic constriction. The mortality in untreated patients approaches 85%.[12]

Study done by Miralda G P showed that in patients with acute pericarditis and with features suggesting associated disease the likelihood of this being the cause for the pericardial syndrome is very high. Therefore, associated disease should be investigated accordingly. In the immunologically competent patient presenting with classically acute pericarditis, the probability of acute idiopathic pericarditis is about 90%, this probability being even greater when clinical features subside in the following few days. No further studies are needed in such cases. In this study, tamponade was significantly more common in patients with tuberculous or purulent pericarditis or neoplasia, but the high prevalence of acute idiopathic pericarditis made it the leading cause in absolute terms. However, in tamponade without findings of inflammation (chest pain or rub) they have demonstrated a likelihood ratio of 3.0 for neoplasia, although other etiologies should be considered as well. Other features such as a sustained clinical course (for example, over three weeks) also increase the likelihood of specific disease. An exception is tamponade with massively haemorrhagic effusion, which is often but not always caused by malignancy. In addition, purulent pericarditis should always be considered in predisposing diseases (such as pleural empyema or mediastinal infection), even in the absence of definite features of pericarditis or tamponade.[13]

Malignancy has been noted to be the most common cause of pericardial effusion in the Western world. Neoplastic pericardial effusion may develop from direct extension or metastatic spread of the underlying malignancy, from an opportunistic infection, or from a complication of radiation therapy or chemotherapeutic toxicity.[14]

CLINICAL FEATURES

In acute pericarditis prodrome of fever, malaise, and myalgia is common, but elderly patients may not be febrile. Major symptoms are

retrosternal or left precordial chest pain (radiates to the trapezius ridge, can be pleuritic or simulate ischemia, and varies with posture) and shortness of breath. The pericardial friction rub can be transient, mono, bi- or triphasic. Pleural effusion may be present. Heart rate is usually rapid & regular.[3]

The clinical presentation of pericardial effusion is varied according to the speed of pericardial fluid accumulation and the aetiology of the effusion with possible symptoms that may be related to the causative disease. The rate of pericardial fluid accumulation is critical for the clinical presentation. If pericardial fluid is quickly accumulating such as due to wounds or iatrogenic perforations, the evolution is dramatic and only small amount of blood are responsible for a quick rise of intrapericardial pressure and overt cardiac tamponade in minutes. On the contrary a slowly accumulating pericardial fluid allows the collection of a large effusion in days to weeks before a significant increase in pericardial pressure becomes responsible of symptoms and signs. Classical symptoms include dyspnoea on exertion progressing to orthopnoea, chest pain, and/or fullness. Additional occasional symptoms due to local compression may include nausea (diaphragm), dysphagia (oesophagus), hoarseness (recurrent laryngeal nerve), and hiccups (phrenic nerve). Non-specific symptoms also include also cough, weakness, fatigue, anorexia and palpitations and reflect the compressive effect of the pericardial fluid on contiguous anatomic structures or reduced blood pressure and secondary sinus tachycardia.

Subacute pericardial tamponade is a difficult diagnosis to make on mere clinical grounds because widely known signs of pericardial tamponade lack sensitivity and specificity.[15] The classical findings of cardiac tamponade have been described by the thoracic surgeon Beck in 1935. Beck identified a triad including hypotension, increased jugular venous pressure, and a small and quiet heart. This triad was classically identified in ‘surgical tamponade’ with acute cardiac tamponade due to intrapericardial haemorrhage because of trauma, myocardial or aortic

rupture. The Beck triad may be lacking in patients with ‘medical tamponade’ with slowly accumulating pericardial fluid. Hypotension is absolute or relative. Acute cardiac tamponade is usually associated with low blood pressure (,90 mmHg) but may be only slightly reduced in subacute, or chronic tamponade. Hypertensive patients may have normal to mildly elevated blood pressure concomitant to cardiac tamponade. Fever is a non-specific sign that may be associated with pericarditis due to either infectious or immune-mediated (i.e. systemic inflammatory diseases) etiology.

On physical examination classic signs include neck vein distention with elevated jugular venous pressure at bedside examination, pulsus paradoxus, and diminished heart sounds on cardiac auscultation. Pericardial friction rubs are rarely reported, they can be usually detected in patients with concomitant pericarditis. Rubs which occur during the maximal movement of the heart within its pericardial sac, are said to be generated by friction between the two inflamed layers of the pericardium. However, this is an oversimplification of the explanation for its mechanism as patients with a pericardial effusion may also have an audible friction rub, and there is no precise correlation between pericardial rubs and size of the effusion, although pericardial rubs may be easier to hear in patients without a pericardial effusion, but this finding is not universal and is not well-documented.

Pulsus paradoxus was described for the first time by Kussmaul in 1873 as a palpable reduction of radial pulse on inspiration in patients with cardiac tamponade. The so-called paradox was the ‘waxing and waning’ of the peripheral pulse, in contrast to the unvarying strength of the apical cardiac impulse. Pulsus paradoxus is classically defined as an inspiratory reduction of at least 10 mmHg of the systolic blood pressure it can be easily detected by recording the systolic pressure at which Korotkoff sounds are first audible and the systolic pressure at which they are audible through the whole respiratory cycle. Pulsus paradoxus is due to exaggerated ventricular

interdependence occurring in cardiac tamponade when overall volume of cardiac chambers becomes fixed and any change in the volume of one side of the heart causes the opposite changes in the other side (i.e. inspiratory increase of venous return and right chambers with decreased left chambers volume and reduced systemic blood pressure). In the absence of cardiac tamponade reported symptoms and signs are non-specific and may include: shortness of breath, dyspnea on exertion, fever, chills, chest pain, paroxysmal nocturnal dyspnoea, orthoponea, cough, and oedema. All these symptoms, and signs may be secondary to the underlying cause of the effusion (i.e. systemic disease and pericarditis) or the initial mechanical interference of pericardial fluid with cardiac function or surrounding anatomical structures [16]. Specific clinical features (fever > 38°C, subacute course, large effusion or tamponade, and aspirin or NSAID failure) may be useful to identify higher risk of specific causal conditions and complications.[17]

INVESTIGATIONS

Electrocardiography

The 12-lead electrocardiogram in patients withacute pericarditis classically shows widespread upward concave ST-segment elevation and PR-segment depression. The electrocardiographic abnormalities may evolve through four phases: diffuse ST-segment elevation and PR-segment depression (stage I); normalization of the ST and PR segments (stage II); widespread T-wave inversions (stage III); and normalization of the T waves (stage IV). Although the prompt institution of therapy may prevent the appearance of all four stages, the changes in stage I are observed in more than 80 percent of patients with pericarditis. ST-segment elevation also occurs in patients with myocardial infarction, but several features may help to distinguish these entities. With infarction, the ST-segment elevations are often convex (domeshaped) rather than concave in configuration, and they are regional rather than widespread; Q-wave formation and loss of R-wave voltage often occur; T-wave inversions appear before the ST

segments return to baseline; PR-segment depression is uncommon; and atrioventricular block or ventricular arrhythmias are common.[10]

In a study by Bruce M A atypical ECG responses include absence of ST deviations, which conceals the diagnosis, and restricted distribution of ST deviations, which suggests myocardial injury. Among 44 consecutive patients with acute pericarditis, 19 (43%) had atypical ECGs. Although all 19 had a pericardial rub, eight had no ST deviations in the limb leads and seven developed no ST changes, including three with no ECG abnormalities of any kind. Patients with typical ECGs by ST segment criteria were more likely to progress to T wave inversion. PR segment deviations occurred in 14 patients with typical, and 14 with atypical ECGs. In four of the latter, the PR segment shifts were the only ECG sign.[18]

Cardiac Biomarkers

Acute pericarditis may be associated with increases in serum biomarkers for myocardial injury, including modest elevations in the MB fraction of creatine kinase (CK-MB) and serum cardiac troponin I (cTnI). Mild increases in cTnI often occur in the absence of elevations in CK-MB. In an unselected population of patients with acute pericarditis this finding has been reported in 32% of cases, but this rate may be as high as 50 to 70% including only hospitalized patients. The rise in serum cTnI in acute pericarditis is transient, typically resolving within 7 to 10 days after presentation, and roughly related to the extent of myocardial inflammation. Associated features include younger age, male gender, ST-segment elevation, pericardial effusion at presentation, and recent onset. Unlike acute coronary syndromes, elevations of cTnI do not seem to carry an adverse prognosis.[19]

Markers of acute myocardial injury may be elevated if myopericarditis is present, but not to the threshold noted for myocardial infarction. The mildly elevated markers have no bearing on the outcome of acute pericarditis.[2]

Inflammatory Markers

Inflammatory markers like erythrocyte sedimentation rate(ESR) and C-reactive protein(CRP) provide little insight into the cause or management of patients with acute pericarditis. CRP may be more sensitive marker that rises earlier and returns to baseline sooner than ESR in cases of pericardial inflammation and resolution of inflammation.[2]

Chest X-Ray

A chest X-ray is generally normal in patients with acute pericarditis since an increased cardiothoracic ratio only occurs with pericardial effusions exceeding 300 ml. In the case of pleuropulmonary diseases, signs of pleuropericardial involvement may be found in patients with pericarditis.[3]

CT or MR

In the evaluation of pericardial disease, computed tomography (CT) and magnetic resonance (MR) imaging traditionally have been used as adjuncts to echocardiography. However, CT and MR imaging are particularly useful as sensitive and noninvasive methods for evaluating loculated or hemorrhagic pericardial effusion, constrictive pericarditis, and pericardial masses. Both CT and MR imaging provide excellent delineation of the pericardial anatomy and can aid in the precise localization and characterization of various pericardial lesions, including effusion, constrictive pericarditis and pericardial thickening, pericardial masses, and congenital anomalies such as partial or complete absence of the pericardium. Both modalities provide a larger field of view than echocardiography, allowing the examination of the entire chest and detection of associated abnormalities in the mediastinum and lungs. Soft-tissue contrast on CT scans and MR images are also superior to that on echocardiograms.[20]

Echocardiography

Echocardiography should be the initial imaging modality used if pericardial effusion is suspected. It is an innocuous technique, highly reliable, and readily available at the patient's bedside. Due to its versatility, echocardiography is the technique of choice to identify hemodynamic compromise in the setting of pericardial effusion. Pericardial effusion appears as an echo-free space between the two layers of the pericardium. Echocardiographic assessment should be structured and focused to (i) differentiate between global or localized effusion; (ii) quantify the effusion; (iii) describe fluid appearance; and (iv) analyze hemodynamic compromise. Standard views along with two-dimensional (2D) echocardiographic, M-Mode, and Doppler analysis should be routinely used in the assessment of pericardial effusion. Four standard views should be used for reproducibility subcostal, four-chamber, and parasternal long and short axis however, in emergent situations, a single view (usually the subcostal one) is enough to diagnose significant pericardial effusion. A combination of the aforementioned four standard views should be used to determine whether the effusion is global or localized.

Pericardial effusion in adults is commonly classified according to the size i.e. mild (<10 mm), moderate (10–20 mm), and severe (>20 mm).[21] The most common and easy view to reproduce is the parasternal long axis, but it is still important to compare with other standard views, such as the parasternal short axis and the four-chamber view. A subcostal view is also very commonly used, but it is important to know that depending on the angle of the probe, it is easy to overestimate the size of the effusion. At end diastole, echo-free space between both pericardial layers can be measured along the posterior wall of the left ventricle (LV). M-mode echocardiography in the parasternal long axis shows an echo-free space throughout between the visceral and the parietal pericardium. This sign is only seen in systole in case of mild effusions and during the complete cycle when the effusion is at least moderate.

A qualitative assessment can be made by defining whether it is global or loculated, transudative, exudative, or containing clotted blood or fibrin. Intrapericardial fibrin and pyogenic exudates can create tabications and loculate the effusion. Particular echogenic densities such as the ones seen in complex exudates or hemopericardium with the presence of fibrin strands and clots are easily recognized by echocardiography. On the other hand, transudates will usually show a more echo-free signal giving a characteristic sinusoid image in M-mode echocardiography that could help differentiate complex exudates from transudates. Hemopericardium can be differentiated because of the spontaneous contrast signal resulting from the swirling of the blood within the pericardial space. Nevertheless, a complete clinical assessment complemented by laboratory tests and imaging studies should always be taken into consideration to find the underlying condition.

In cardiac tamponade the primary abnormality is compression of all cardiac chambers due to fluid accumulation in the intrapericardial space, resulting in increased intrapericardial pressure sufficient to raise the pressure surrounding the heart to the point where cardiac filling is altered. Abnormal right heart filling is the earliest sign of a hemodynamically significant pericardial effusion.

The inspiratory exaggerated diastolic collapse of the free walls of the right atrium and/or right ventricle due to compression of these relatively low-pressure structures is the hallmark. Right atrial collapse, especially when it persists for more than a third of the cardiac cycle, is highly sensitive and specific for tamponade(sensitivity:100%; specificity:82%), but brief right atrial collapse can occur in the absence of tamponade. Doppler echocardiography helps to identify the hemodynamic significance of tamponade and reflects the increased ventricular interdependence between the left & right ventricles. Tricuspid flow increases and mitral flow decreases during inspiration (reverse in expiration). Systolic and diastolic flows are reduced in

systemic veins in expiration and increase of reverse venous flow is seen during atrial systole.[2,22]

In constrictive pericarditis, early diastolic mitral inflow is reduced with the onset of inspiration, and isovolumic relaxation time is prolonged. With expiration, mitral inflow returns to normal, and isovolumic relaxation time shortens. Typically, patients with CP demonstrate an increase in early diastolic mitral inflow velocity of ≥25% during expiration compared with inspiration. These changes can be seen with the first beat of inspiration, when a decrease in transmitral flow velocity is noted; the reverse occurs with expiration. Sensitivities and specificities of Doppler assessment of respiratory changes have been reported to be as high as 85% to 90%. Importantly, the percentage of respiratory change in patients with “mixed” constrictive and restrictive disease (eg, mediastinal radiation) is markedly lower. After complete pericardiectomy mitral inflow patterns return to normal, and little respiratory variation is seen on Doppler echocardiography. Accordingly, Doppler assessment of respiratory variation has been reported to be useful for evaluating the outcome of pericardiectomy. Patients who had minimal respiratory variation after pericardiectomy were asymptomatic compared with those who continued to show respiratory variation.[23]

TREATMENT

If a specific cause of pericarditis is identified, treatment should be directed accordingly. For patients with idiopathic pericarditis, therapy is aimed at the relief of chest pain and inflammation. However, such treatment does nothing to prevent tamponade, constriction, or recurrent pericarditis. According to observational studies, Non-steroidal anti-inflammatory agents (NSAIDs) are the mainstay of therapy, they are effective in relieving chest pain in 85 to 90 percent of patients, and limited observational data suggest that the various agents have similar efficacy. Aspirin (2 to 4 g daily), indomethacin (75 to 225 mg daily), and ibuprofen (1600 to 3200 mg daily) are prescribed most often, with

ibuprofen being the preferred drug, because it has a lower incidence of adverse effects than the others. Aspirin is preferable for patients who have had a recent myocardial infarction, since other NSAIDs impair scar formation according to animal studies. Indomethacin should be avoided in patients with coronary artery disease, since it diminishes coronary blood flow.[10,24]

Adler Y et al concluded that colchicine may be an extremely promising adjunct to conventional treatment and may ultimately serve as the initial mode of treatment, especially in idiopathic cases of acute pericarditis.[25]

The treatment of cardiac tamponade is drainage of the pericardial effusion. Medical management is usually ineffective and should be used only while arrangements are made for pericardial drainage. Fluid resuscitation may be of transient benefit if the patient is volume depleted (hypovolemic cardiac tamponade). The use of inotropic agents is usually ineffective because there is already intense endogenous adrenergic stimulation. The initiation of mechanical ventilation in a patient with tamponade may produce a sudden drop in blood pressure because the positive intra-thoracic pressure will contribute to a further impairment of cardiac filling.[1,26]

In the absence of clinical evidence of tamponade, echocardiographic findings of right-sided diastolic collapse do not mandate emergency pericardiocentesis. For example, emergency pericardial drainage is not recommended in a patient who has a nontraumatic pericardial effusion with right-sided collapse if there is an adequate stable blood pressure (>110 mm Hg systolic) without a paradoxical pulse (ie, ≤10 mm respiratory variation in systolic pressure). However, the patient must be observed carefully because the development of only a small additional amount of pericardial fluid can result in tamponade. In some patients, the echocardiographic signs of cardiac compression will resolve within a few days, and pericardiocentesis can be avoided if there is no other indication.

Traditionally, nonemergent pericardiocentesis has been performed in the cardiac catheterization laboratory under fluoroscopic guidance with invasive hemodynamic monitoring. Performing pericardiocentesis in this setting provides the option of utilizing right heart catheterization before and after the procedure to confirm the diagnosis, if necessary, and to detect effusive-constrictive pericardial disease. More recently, echocardiographic-guided pericardiocentesis has been demonstrated to be a safe and effective procedure that can be performed at the bedside. During this procedure the ideal entry site (minimal distance from skin to pericardial fluid without intervening structures) can be defined. Continued drainage of the pericardial fluid through an indwelling catheter minimizes the risk of reoccurrence of the effusion. If pericardial tissue is required for diagnosis or in the case of purulent pericarditis or recurrent effusions, surgical drainage may be the preferred treatment. Surgery is also the treatment for traumatic hemopericardium.

Surgical drainage of a pericardial effusion is usually performed through a limited subxiphoid incision. This allows direct visualization and biopsy of the pericardium. The diagnosis accuracy can be improved by inserting a pericardioscope. This provides direct visualization of a much larger area of the pericardium and the ability to obtain multiple biopsies. Recently, a flexible pericardioscope has been developed that can be inserted percutaneously.

Malignant pericardial effusions can frequently reoccur. Such recurrent pericardial effusions may necessitate the surgical creation of a pericardial window that allows the effusion to drain into the pleural space, preventing reoccurrence of cardiac tamponade. An attractive alternative in these patients, especially if their overall prognosis is poor from the malignancy, is the percutaneous creation of a pericardial window by balloon dilation.

The symptoms and constrictive features may resolve with medical therapy alone in patients with relatively acute onset pericardial

constriction. In chronic pericardial constriction, definitive treatment is surgical pericardial decortication, widely resecting both the visceral and parietal pericardium.[27] This operation is a major undertaking with substantial risk (6% mortality even in the most experienced centers). In some patients, it does not immediately restore normal cardiac function, which may require some time after removal of the constricting pericardium to return to normal. The largest surgical series from the Mayo Clinic and the Cleveland Clinic indicate that patients with constriction due to idiopathic or viral pericarditis do best and patients with radiation-induced constriction fare most poorly after surgery.[1]

MATERIALS AND METHODS

This study was conducted in a tertiary care hospital. This was an observational study and was conducted over a period of one and a half year from January 2020 to June 2021. All patients hospitalized between this period with the confirmed diagnosis of pericardial diseases were included in the study.

SOURCE OF DATA

All patients admitted in Medicine/Cardiology department of DMCH with diagnosis of pericardial diseases (confirmed by ECHO / ECG / Clinical findings) were included in the study. Further, these patients were included based on the following criteria's.

INCLUSION CRITERIA

- All patients age above 18 years
- All patients with findings suggestive of pericardial diseases (acute pericarditis, pericardial effusion, cardiac tamponade, chronic pericarditis, constrictive pericarditis, recurrent pericarditis, etc) were included.

EXCLUSION CRITERIA

- All patients less than 18 years.
- Post CABG and post ICR (intra-cardiac repair) patients with post-operative effusions in primary hospital admission.

DURATION OF STUDY: It was a time bound study and was conducted over a period of one and a half year from January 2020 to June 2021. A total of 170 patients admitted in Medicine / Cardiology department of DMCH were included in the study.

METHOD OF COLLECTION OF DATA

All the IPD patients fulfilling the inclusion criteria were included in this study. A pre-structured performa was used to record the specific details of these patients. It included the study of their demographical profile (age/gender/co-morbidities), history, clinical findings, lab investigations, ECG findings, 2D ECHO findings, pericardial fluid analysis (if indicated), radiological findings, basic diagnosis. The treatment modalities used were according to the discretion of the treating physician.

A total of 170 hospitalized patients diagnosed with various pericardial diseases were evaluated at our hospital over a period of 1.5 years. The diagnostic criterias used to establish different pericardial diseases are as follows –

1) ACUTE PERICARDITIS

The diagnosis of acute pericarditis is made with presence of at least 2 of the 4 following criteria:(1) pericarditic chest pain (2) pericardial rubs (3) new widespread ST-elevation or PR depression on ECG (4) pericardial effusion (new or worsening).[28]

2) PERICARDIAL EFFUSION

Standard two-dimensional echocardiographic criteria were used to diagnose pericardial effusion and tamponade. According to the size of the echo-free region, pericardial effusion was classified as mild (<10mm), moderate (10–20 mm) or severe (>20 mm).[28]

Evaluation for the cause of pericardial effusion included hemogram, renal function tests, ESR, Mantoux test, thyroid profile, antinuclear antibodies (ANA) test, rheumatoid factor test. Radiological investigations included chest x-ray and chest computed tomography (or magnetic resonance imaging if CT was contraindicated). Pericardial fluid was analyzed for Cells, proteins, lactate dehydrogenase (LDH),

malignant cytology, ADA, gram staining, acid-fast bacillus (AFB) staining, PCR (for M. tuberculosis) and cultures. Clinical history, examination, and specific laboratory investigations for tuberculosis, hypothyroidism, malignancy, uremia, and collagen vascular disease were used to make the final diagnosis of pericardial effusion. To diagnose tubercular pericardial effusion presence of lymphocytic predominance and a positive ADA (cut off value >40UL with sensitivity and specificity of 88% and 83%, respectively) [29] or TB PCR/CB NAAT, as well as active tuberculosis elsewhere in the body, were used as diagnostic markers.

Analyses of the pericardial fluid specific gravity (>1015), protein level (>3.0 g/dL), fluid/serum ratio >0.5, LDH >200 mg/dL, serum/fluid >0.6, and glucose can separate exudates from transudates, but are not directly diagnostic. [16,30]

3) CARDIAC TAMPONADE

The diagnosis is confirmed by an echocardiographic demonstration of moderate or large circumferential pericardial effusion and in most instances, of right atrial compression, abnormal respiratory variation in right and left ventricular dimensions, and in tricuspid and mitral valve flow velocities usually associated with inferior vena cava plethora.

4) CONSTRICTIVE PERICARDITIS

M-mode and two-dimensional transthoracic and Doppler echocardiography are primary imaging modalities in the evaluation of constrictive pericarditis. Major findings include:[28]

- Septal bounce.
- Pericardial thickening and calcifications
- Respiratory variation of the mitral peak E velocity of >25% and variation in the pulmonary venous peak D flow velocity of >20%
- Colour M-mode flow propagation velocity (Vp) >45 cm/sec.
- Tissue Doppler: peak e' >8.0 cm/s.

STATISTICAL ANALYSIS: The data was compiled and analysed by using relevant statistical method. Data was described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATION AND RESULTS

A total number of 170 patients were enrolled in this study. We analyzed the demographical, clinical, radiological parameters of these patients.

DEMOGRAPHIC FEATURES OF STUDY POPULATION

Table 2- DISTRIBUTION OF PATIENTS ACCORDING TO AGE

Age group(years)	No. of cases	Percentage
< 40	35	20.6%
41-60	68	40.0%
> 60	67	39.4%
Total	170	100.0%

Most patients (n=68, 40%) in our study were in the age group between 41-60 years. There were 35(20.6%) patients below the age of 40 years and 67(39.4%) patients above the age of 60 years.

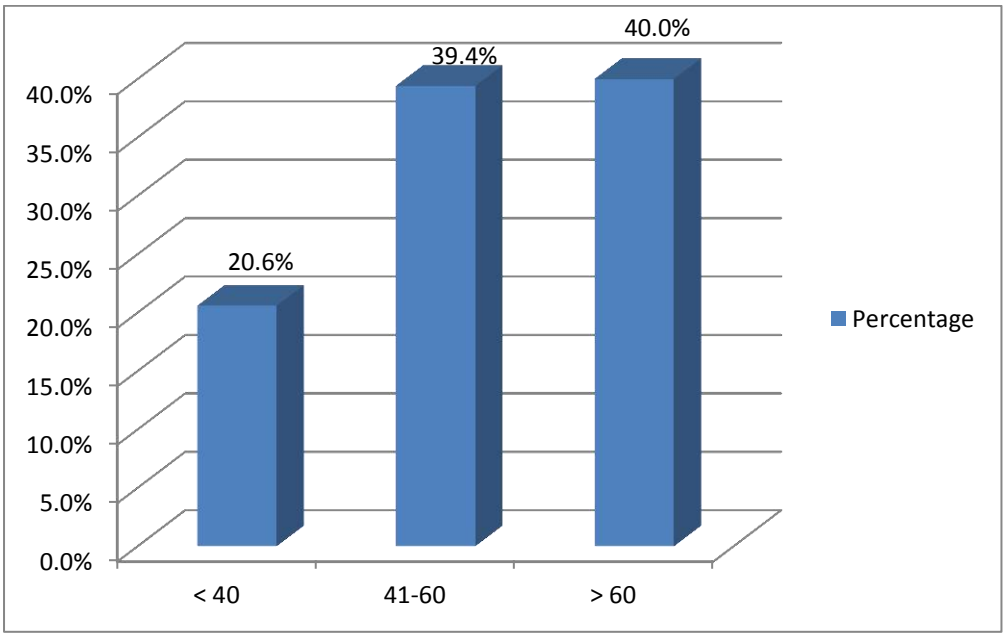

Figure 3. DISTRIBUTION OF PATIENTS ACCORDING TO AGE

Table 3. DISTRIBUTION OF PATIENTS ACCORDING TO GENDER

GENDER	No. of cases	Percentage
Female	70	41.2%
Male	100	58.8%
Total	170	100.0%

In our study, there were 100 males (58.8%) and 70 females (41.2%).

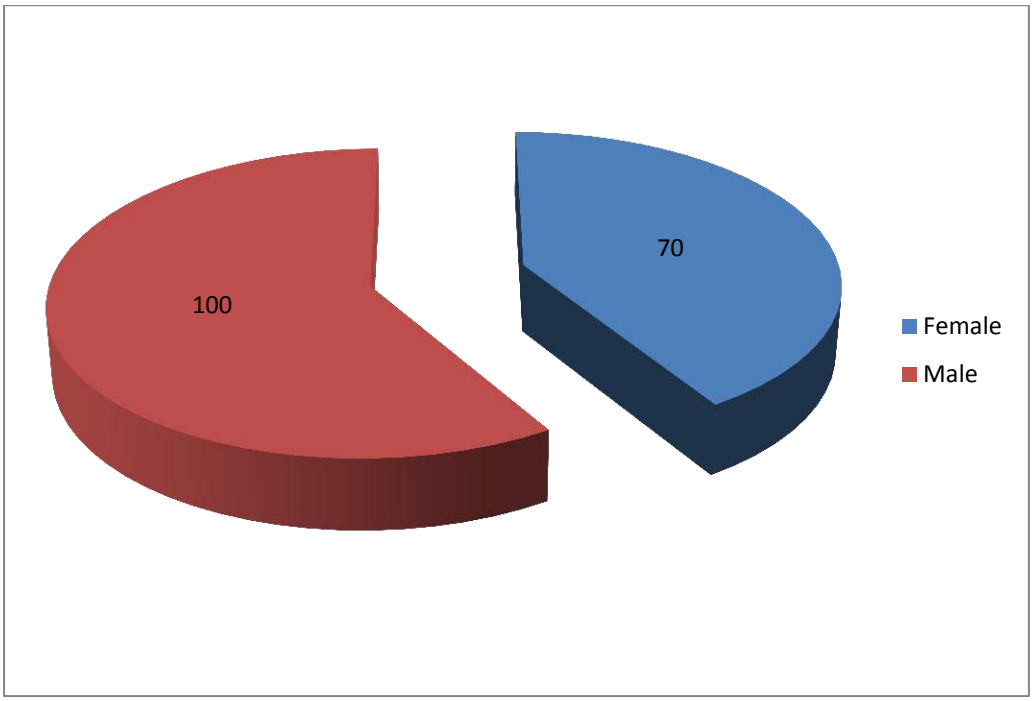

Figure 4.DISTRIBUTION OF PATIENTS ACCORDING TO GENDER

TABLE 4. DISTRIBUTION OF PATIENTS ACCORDING TO VARIOUS CO-MORBIDITIES

MEDICAL CO-MORBIDITIES	No. of cases	Percentage
DM	68	40.0%
HTN	64	37.6%
CKD	34	20.0%
OTHER	28	16.5%
HYPOTHYROIDISM	19	11.2%
IHD	11	6.5%
AUTOIMMUNE	8	4.7%
COPD	6	3.5%
MALIGNANCY	5	2.9%
TB	3	1.8%

In our study, the common co-morbidities seen were diabetes mellitus (n=68, 40%), hypertension(n=64,37.6%) and CKD (n=34,20%) followed by hypothyroidism (n=19,11.2%), IHD(n=11,6.5%), autoimmune (n=8,4.7%), COPD (n=6,3.5%), malignancy (n=5,2.9%), and TB (n=3,1.8%).

Other co-morbidities like RHD, CLD, HCV, RA, etc. were present in about 16.5 % (n=28) cases.

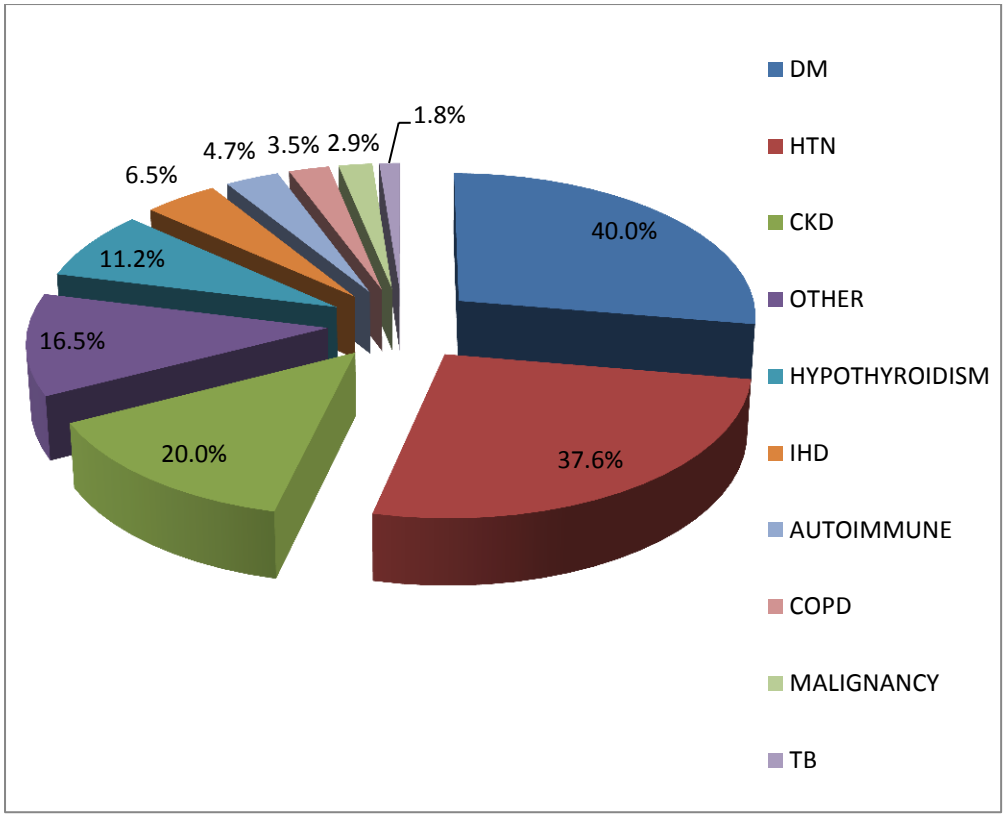

FIGURE 5. DISTRIBUTION OF PATIENTS ACCORDING TO VARIOUS CO-MORBIDITIES

TABLE 5: SPECTRUM OF PERICARDIAL DISEASES OBSERVED

Final Diagnosis	No. of cases	Percentage
ACUTE PERICARDITIS	33	19.4%
CONSTRICTIVE PERICARDITIS	3	1.8%
EFFUSIVE-CONSTRICTIVE	12	7.1%
PERICARDIAL EFFUSION	122	71.8%
Total	170	100.0%

In our study, most of the cases observed were of pericardial effusion (n=122, 71.8%) followed by acute pericarditis (n=33, 19.4%), effusive-constrictive pericarditis (n=12, 7.1%) and constrictive pericarditis (n=3, 1.8%) in that order.

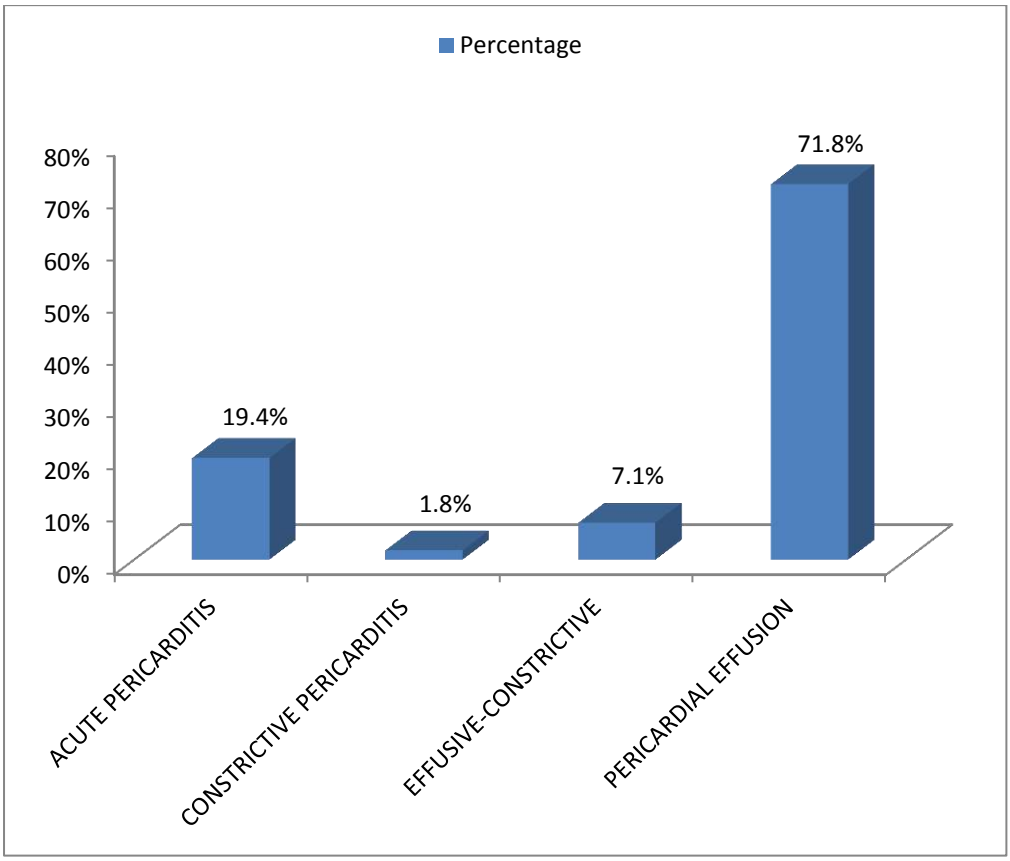

FIGURE 6. SPECTRUM OF PERICARDIAL DISEASES.

Table 6. DISTRIBUTION OF PATIENTS ACCORDING TO VARIOUS PRESENTING SYMPTOMS

CHIEF COMPLAINTS	No. of cases	Percentage
SOB	113	66.5%
CHEST PAIN	39	22.9%
FEVER	34	20.0%
OTHER	66	38.8%

In our study, most common presenting symptoms were shortness of breath (66.5%), chest pain (22.9%) and fever (20%). Other symptoms including cough, generalized body weakness, pedal edema, decreased urine output, palpitations, pain abdomen, etc were present in 38.8% of total patients.

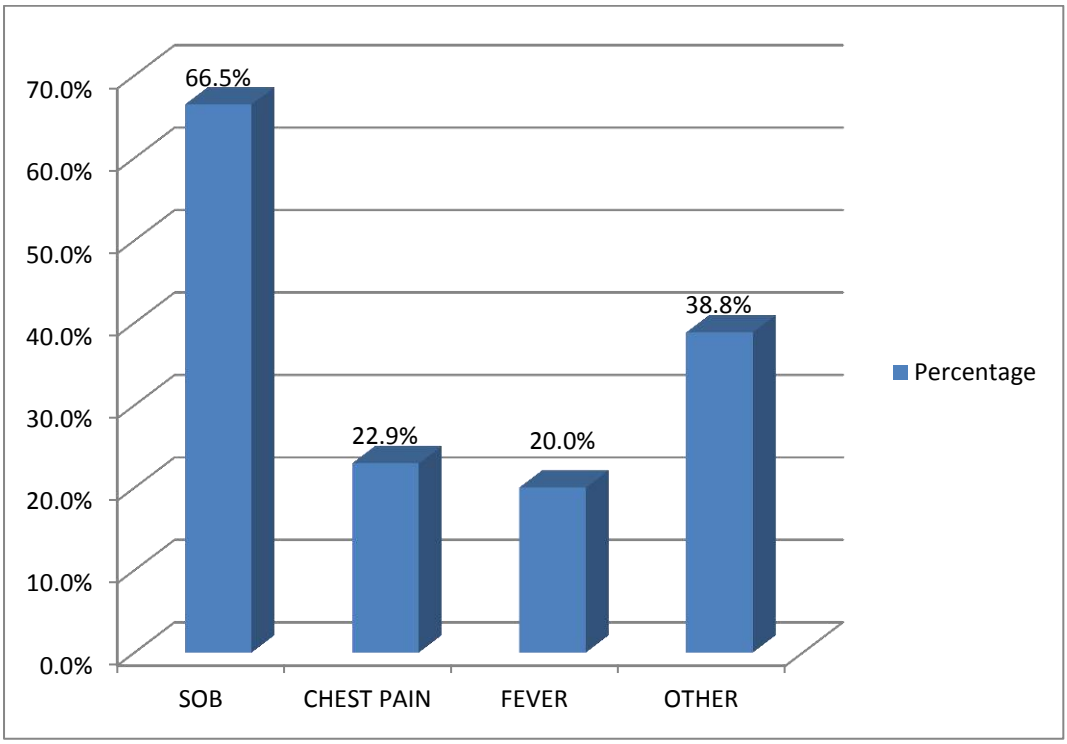

Figure 7. DISTRIBUTION OF PATIENTS ACCORDING TO VARIOUS PRESENTING SYMPTOMS

TABLE 7. SIGNS OBSERVED IN CASES OF PERICARDIAL DISEASES

PHYSICAL SIGNS	NO. OF PATIENTS
RAISED JVP	70
PERICARDIAL RUB	23
PULSUS PARADOX	17
LYMPHADENOPATHY	9
PERICARDIAL KNOCK	2
HYPOTENSION	20
TACHYCARDIA	81

In our study, the most common sign observed on physical examination was tachycardia(n=81,47%) followed by raised JVP(n=70,41%), pericardial rub (n=23,13%), hypotension (n=20,11.7%), pulsus paradoxus (n=17,10%), lymphadenopathy (n=9,5%) and pericardial knock (n=2,1.1%).

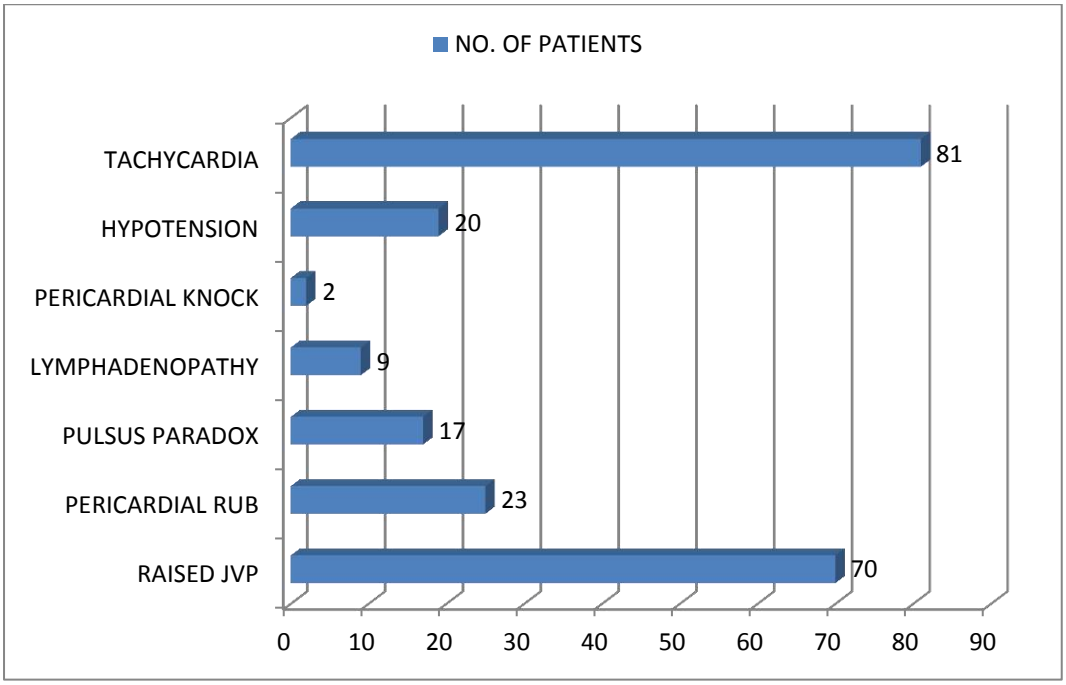

FIGURE 8: SIGNS OBSERVED IN CASES OF PERICARDIAL DISEASES.

TABLE 8. ETIOLOGIES OF PERICARDIAL DISEASES

ETIOLOGY	No. of cases	Percentage
TUBERCULAR	31	18.2%
IDIOPATHIC	30	17.6%
UREMIC	24	14.1%
POST MI	21	12.4%
HFrEF	20	11.7%
MALIGNANCY	18	10.6%
AUTOIMMUNE	7	4.1%
TROPICAL FEVER	6	3.5%
HYPOTHYROIDISM	5	3.0%
HFpEF	4	2.4%
PYOGENIC	4	2.4%

In our study, the most common etiology of pericardial disease was tuberculosis(18.2%) followed by idiopathic (17.6%), uremic(14.1%), post MI (12.4%), HFrEF (11.7%), malignancy (10.6%), autoimmune diseases(4.1%), tropical fever(3.5%), hypothyroidism(3%), HFpEF (2.4%), and pyogenic(2.4%).

Among cases of tropical fever mostly dengue fever cases (n=5,2.9%) were observed.

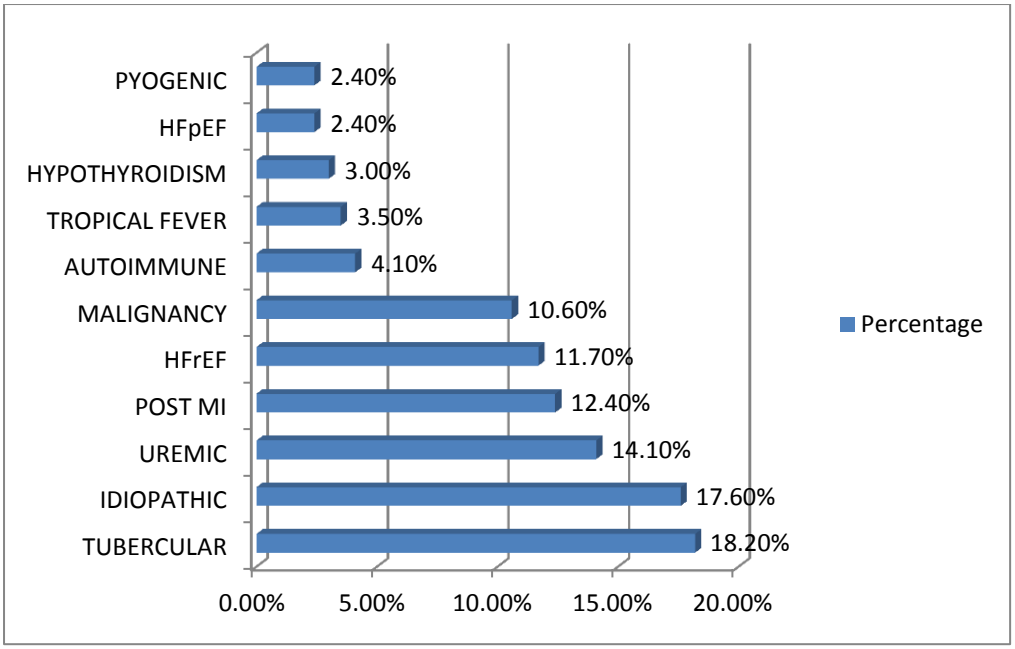

FIGURE 9: ETIOLOGIES OF PERICARDIAL DISEASES IN OUR STUDY

TABLE 9: MALIGNANT ETIOLOGIES IN CASES OF PERICARDIAL DISEASES

MALIGNANCY	No. of cases	Percentage
BREAST CARCINOMA	5	2.9%
ADENOCARCINOMA LUNG	4	2.4%
NHL	2	1.2%
AML	1	0.6%
NEUROENDOCRINE TUMOUR	1	0.6%
PAPILLARY SEROUS CARCINOMA	1	0.6%
SCC LUNG	1	0.6%
BRONCHOGENIC CARCINOMA	1	0.6%
GERM CELL TUMOUR	1	0.6%
GIST	1	0.6%

In our study, the most common malignant etiology seen is breast carcinoma (n=5,2.9%) followed by adenocarcinoma lung(n=4,2.4%) and Non-hodgkin lymphoma(n=2,1.2%). The remaining malignant etiologies included acute myeloid leukemia, neuroendocrine tumour, papillary serous carcinoma, squamous cell carcinoma lung, bronchogenic carcinoma, germ cell tumour, and GIST, one case each.

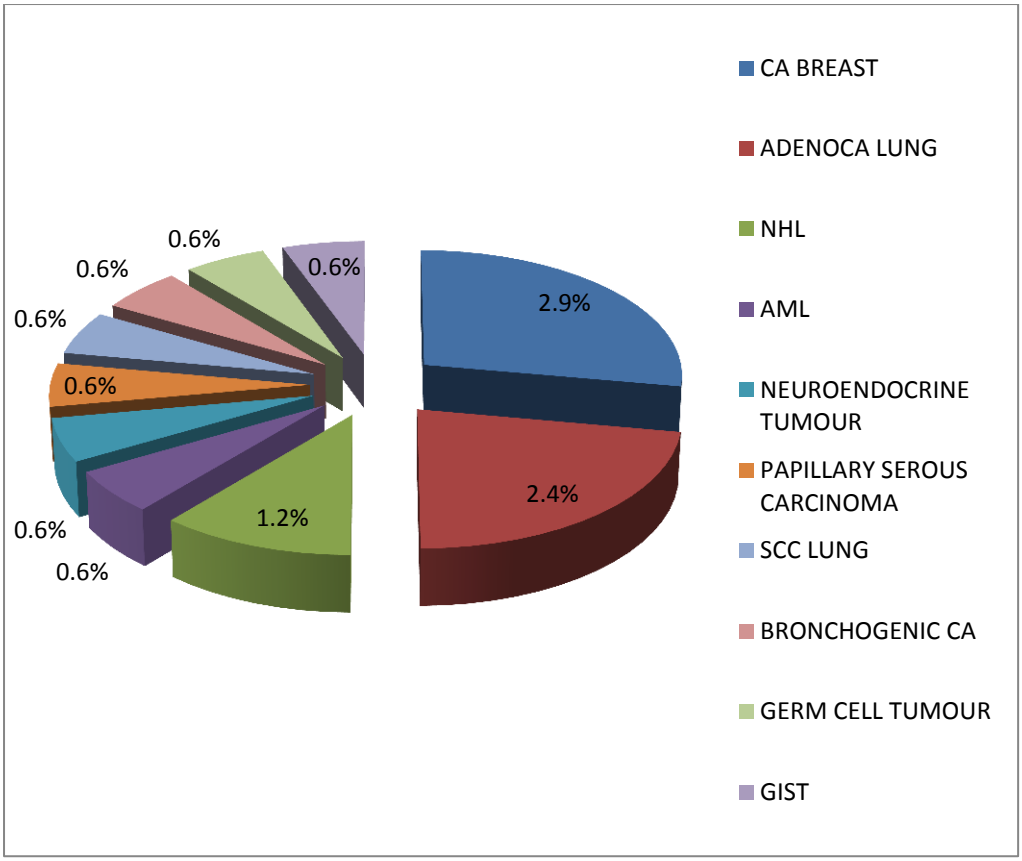

FIGURE 10: MALIGNANT ETIOLOGIES IN CASES OF PERICARDIAL DISEASES

TABLE 10. AUTOIMMUNE ETIOLOGIES IN PERICARDIAL DISEASES

AUTOIMMUNE	No. of cases	Percentage
SLE	5	2.9%
RA/SLE OVERLAP SYNDROME	1	0.6%
SJOGRENS	1	0.6%

In our study, autoimmune etiology included mostly cases of SLE (n=5,2.9%) followed by RA-SLE overlap syndrome(n=1,0.6%) and Sjogrens's syndrome(n=1,0.6%).

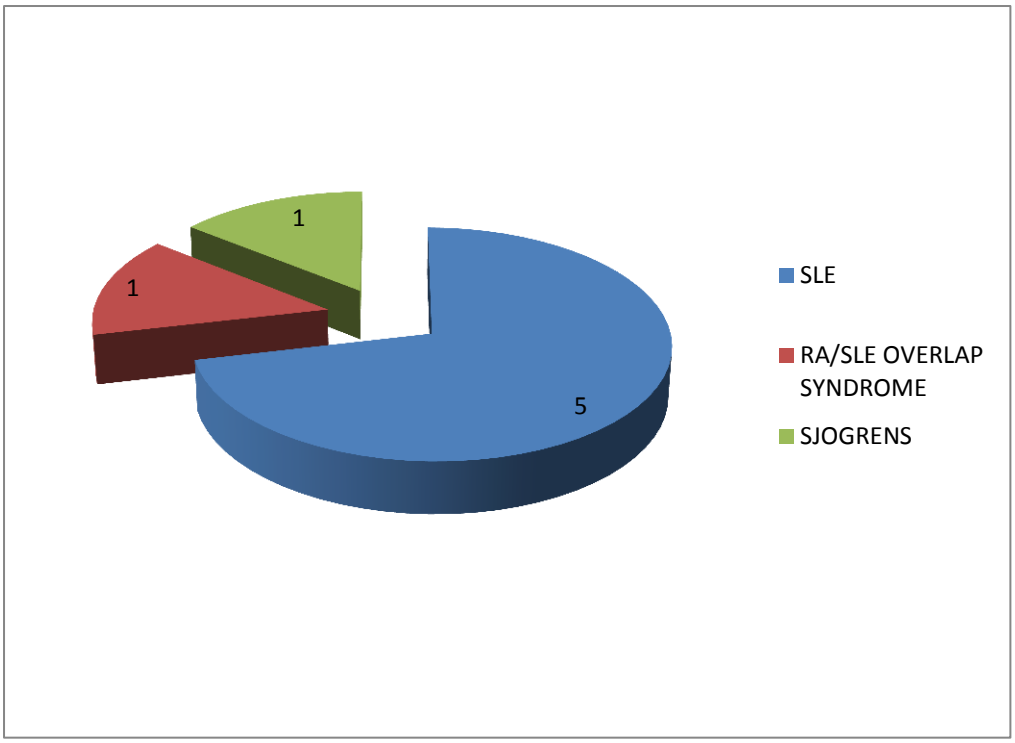

FIGURE 11: AUTOIMMUNE ETIOLOGIES IN PERICARDIAL DISEASES

VARIOUS INVESTIGATION RESULTS IN PERICARDIAL DISEASES

TABLE 11. ECG CHANGES SEEN IN CASES OF PERICARDIAL DISEASES

ECG FINDINGS	No. of cases	Percentage
LOW VOLTAGE ECG	41	24.1%
ST SEGMENT ELEVATION	37	21.7%
OTHERS	25	14.7%
PR SEGMENT CHANGES	6	3.5%
T WAVE LOW/ FLATTENED/ INVERTED	15	8.8%
ELECTRICAL ALTERNANS	2	1.2%

In our study, most common ECG changes found were low voltage QRS complex (n=41,24.1%) followed by ST segment elevation(n=39,21.7%), T- wave changes (n=15,8.8%), PR segment depression (n=6, 3.5%) and electrical alternans (n=2,1.2%). Other ecg abnormalities present were atrial fibrillation, LBBB, RBBB, AVNRT, complete heart block and supraventricular tachycardia in about 14.7%(n=25) cases.

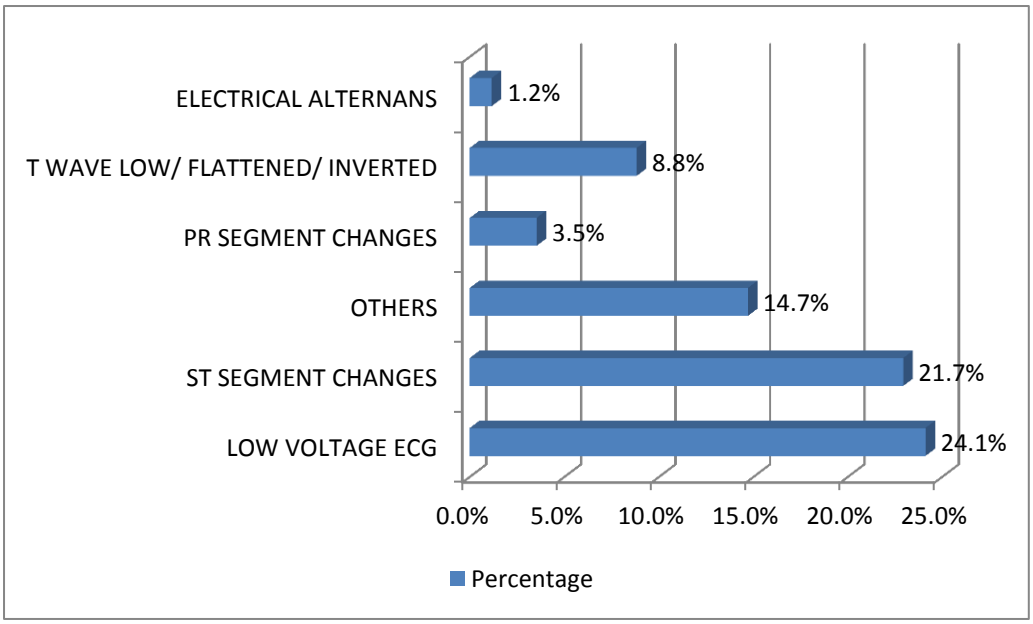

FIGURE 12: ECG CHANGES SEEN IN CASES OF PERICARDIAL DISEASES

TABLE 12. LABORATORY PARAMETERS IN VARIOUS PERICARDIAL DISEASES

LAB PARAMETER	ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)		Total
LEUCOCYTOSIS (TLC> 10)	22	66.67%	2	66.67%	5	41.67%	58	47.54%	87
ESR(>20)	24	72.73%	2	66.67%	7	58.33%	89	72.95%	122
CRP(>6)	7	21.21%	2	66.67%	4	33.33%	23	18.85%	36
TROP-T +	4	12.12%	0	0.00%	1	8.33%	18	14.75%	23

In our study, leukocytosis was seen in 51.1% (n=87) cases, raised ESR in 71.7%(n=122) cases, raised CRP in 21.1%(n=36) cases and positive quantitative troponin-T was seen in 13%(n=23) cases.

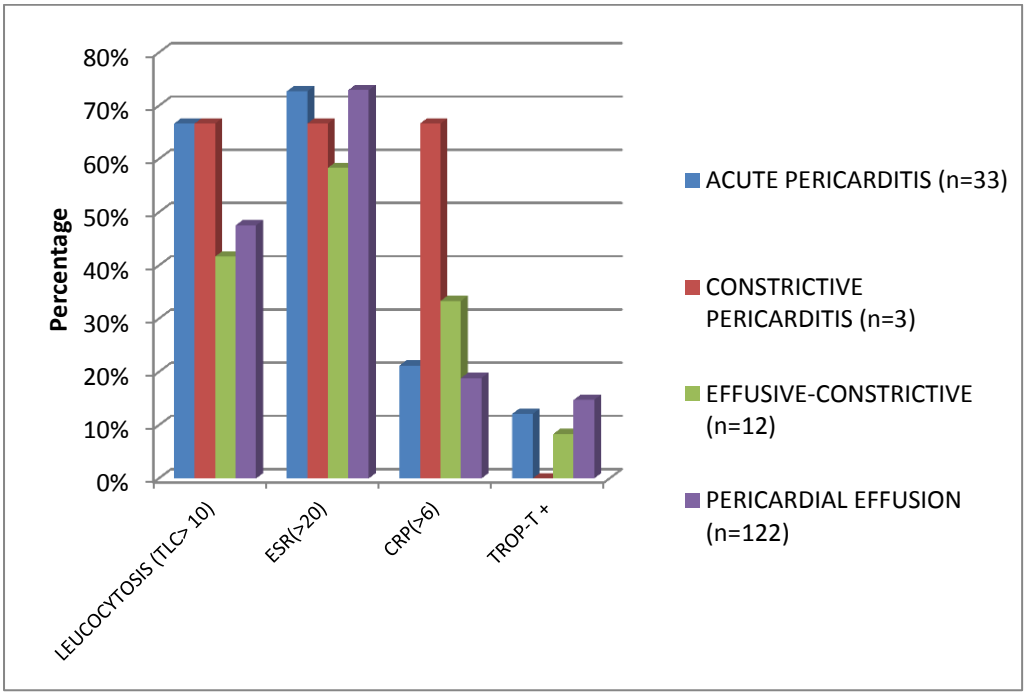

FIGURE 13. LABORATORY PARAMETERS OBSERVED IN OUR STUDY

TRANSTHORACIC ECHOCARDIOGRAPHIC FINDINGS IN CASES OF PERICARDIAL DISEASES

TABLE 13: SPECIFIC ECHOCARDIOGRAPHIC FINDINGS IN PERICARDIAL DISEASE CASES

	No. of cases	Percentage
THICKENED PERICARDIUM	6	3.5%
EXUDATIVE STRANDS/FRONDS	22	12.9%
EFFUSIVE-CONSTRICTIVE	12	7.1%
CARDIAC TAMPONADE	20	11.8%
RESPIRATORY VARIATION OF MV & TV VELOCITIES	17	10.0%

In our study, specific ECHO findings reported were exudative strands/fronds (12.9%), cardiac tamponade physiology(11.8%), respiratory variation of mitral & tricuspid valve velocities(10.0%), effusive-constrictive physiology(7.1%) and pericardial thickening(3.5%) Annulus paradoxus was reported in 2 cases of constrictive pericarditis and 1 case of effusive-constrictive pericarditis.

In our study pericardial thickening was reported in 2 cases (66.7%) of constrictive pericarditis, 2 cases(16.67%) of effusive –constrictive pericarditis and in 2 cases(1.64%) of pericardial effusion.

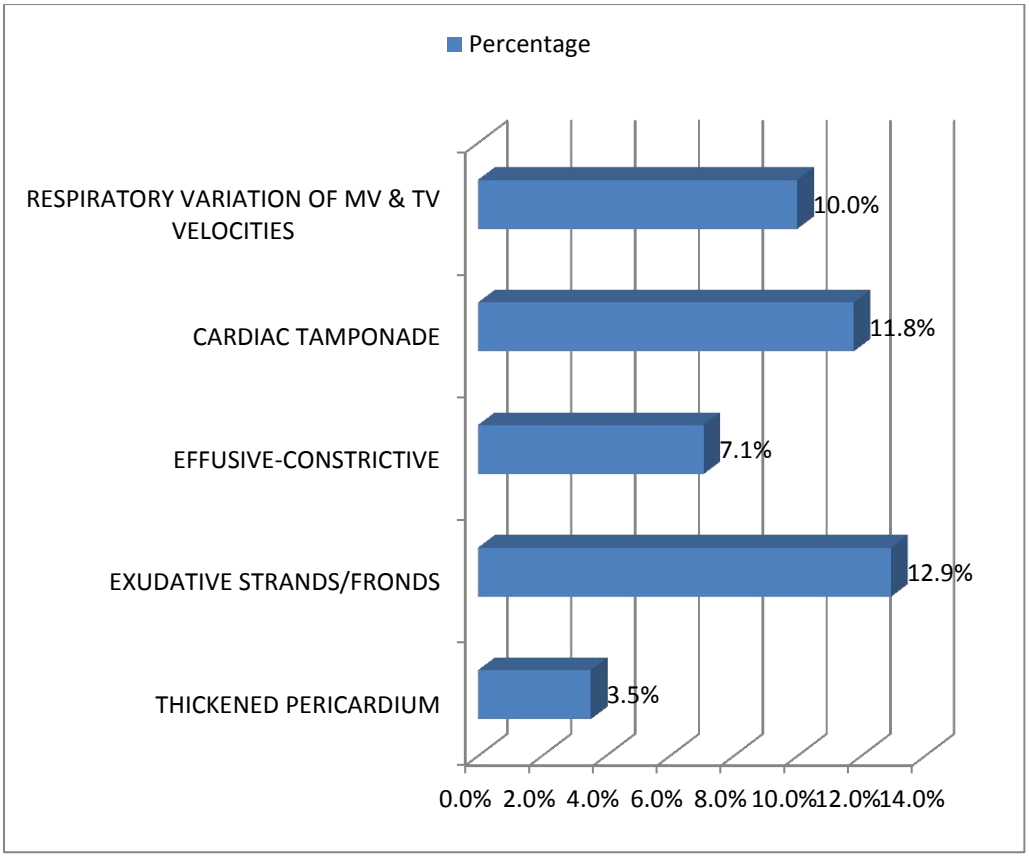

FIGURE 14: SPECIFIC ECHO FINDINGS IN PERICARDIAL DISEASE CASES

TABLE 14: PERICARDIAL EFFUSION OBSERVED ON ECHO

PERICARDIAL EFFUSION	No. of cases	Percentage
MILD	90	54.2%
MODERATE	51	30.0%
SEVERE	25	15.1%
Total	166	100.0%

In our study, out of total 170 cases, pericardial effusion was reported in 166 cases. Acute pericarditis cases with mild or moderate effusions are also included. In 4 cases of acute pericarditis, pericardial effusion was not observed. 54.2% (n=90) had mild pericardial effusion, 30% (n=51) had moderate pericardial effusion and 15.1% (n=25) cases had severe pericardial effusion.

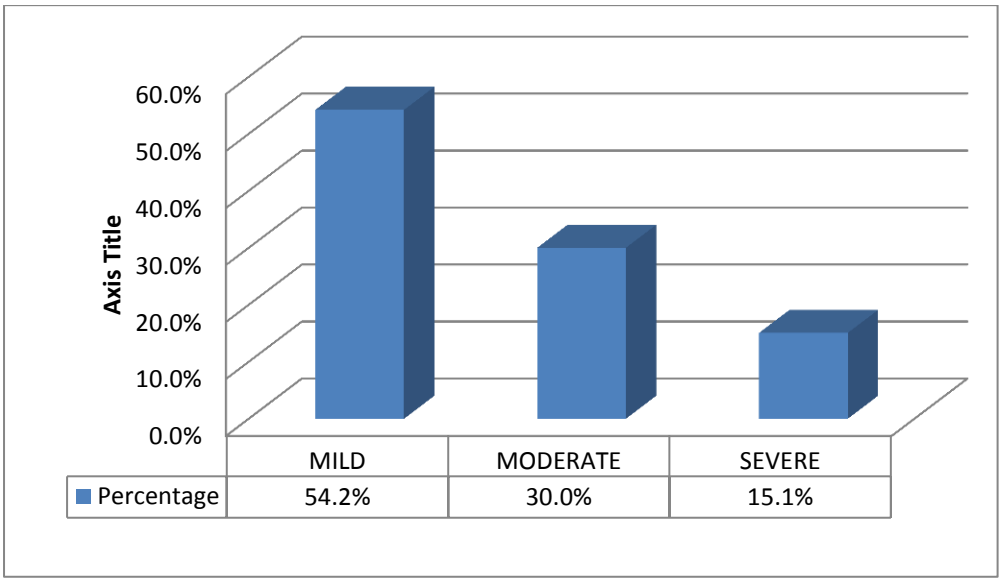

FIGURE 15: DEGREE OF PERICARDIAL EFFUSION SEEN IN ECHO.

TABLE 15: CASES ASSOCIATED WITH SEVERE LEFT VENTRICULAR SYSTOLIC DYSFUNCTION

LVEF	ACUTE PERICARDITIS (n=33)		PERICARDIAL EFFUSION (n=122)		Total
< 40%	16	48.48%	43	35.24%	59
> 40%	17	51.52%	79	64.75%	96

In our study, 16 (48.48%) out of 33 patients of acute pericarditis were found to have myocardial involvement. Among cases of pericardial effusion 43 patients (35.2%) were found to have severe left ventricular dysfunction.

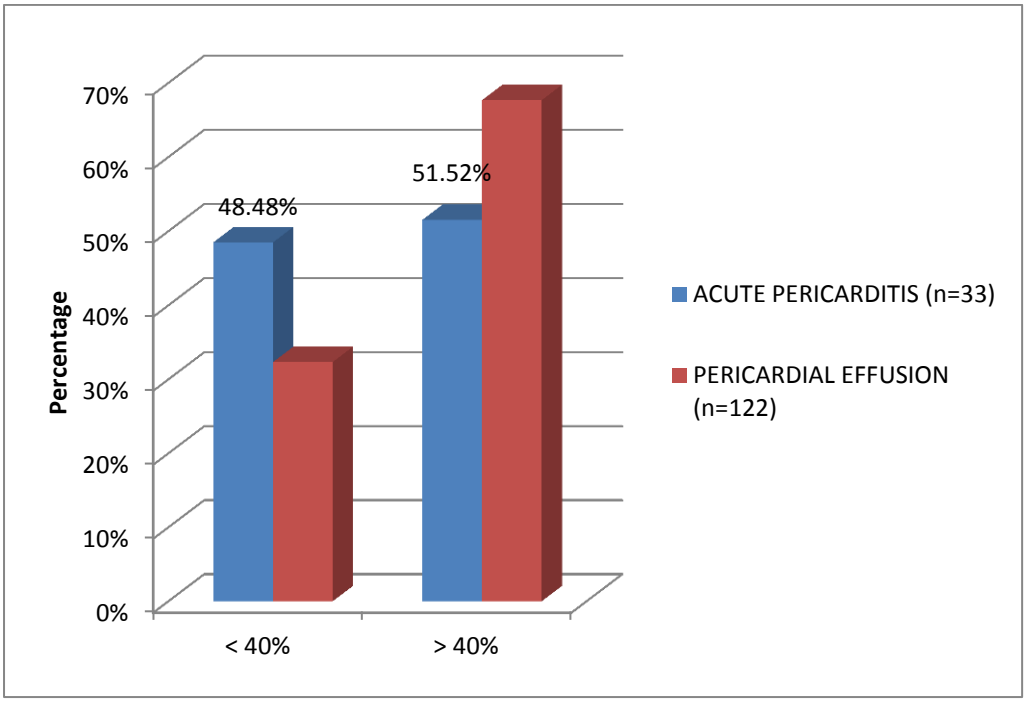

FIGURE 16: ASSOCIATED LEFT VENTRICULAR SYSTOLIC DYSFUNCTION IN PERICARDIAL DISEASES

TABLE 16: EXUDATIVE STRANDS/FRONDS ON ECHO IN CASES OF PERICARDIAL DISEASES

EXUDATIVE STRANDS/ FRONDS	ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)		Total
ABSENT	32	96.97%	3	100.00%	7	58.33%	106	86.89%	148
PRESENT	1	3.03%	0	0.00%	5	41.67%	16	13.11%	22

Exudative strands/fronds were seen in 16(13.1%) patients of pericardial effusion, 5(41.7%) patients with effusive-constrictive pericarditis and in 1(3%) patient with acute pericarditis. Most common etiology observed was tubercular (n=7) followed by pyogenic(n=3) and malignancy(n=2).

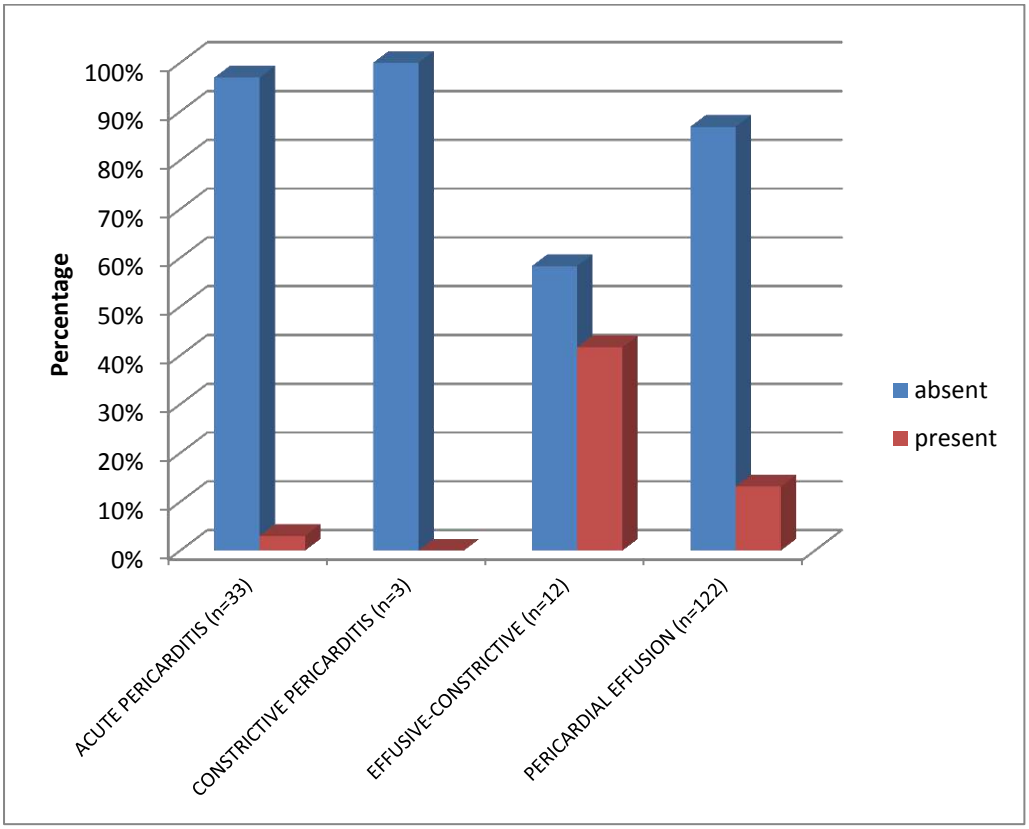

FIGURE 17: EXUDATIVE STRANDS/FRONDS ON 2DECHO IN CASES OF PERICARDIAL DISEASES

TABLE 17. RESPIRATORY VARIATION OF MV & TV VELOCITIES SEEN ON ECHO

RESPIRATORY VARIATION OF MV & TV VELOCITIES	ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)		Total
ABSENT	16	48.4%	0	0.0%	4	33.3%	116	95%	153
PRESENT	0	0.0%	3	100%	8	66.6%	6	4.9%	17

In our study, respiratory variation of TV & MV velocities were observed in 3 patients (100%) of constrictive-pericarditis, 8 patients(66.6%) of effusive-constrictive pericarditis and in 6 patients(4.9%) with pericardial effusion , proving its high sensitivity in cases of constrictive & effusive-constrictive pericarditis.

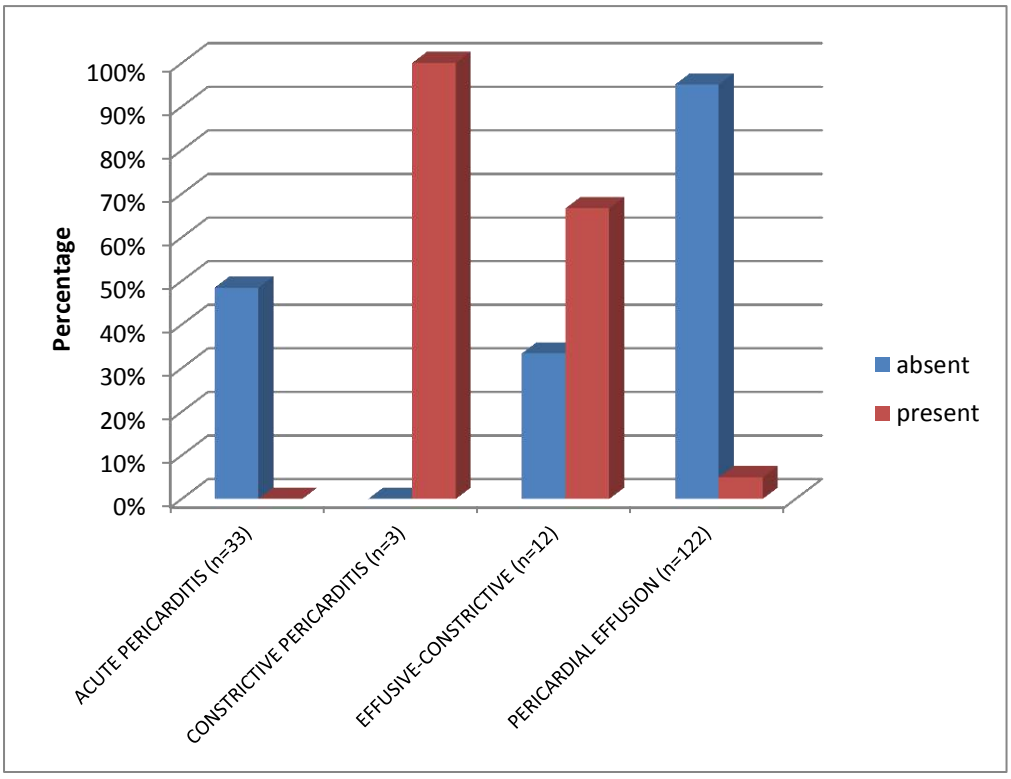

FIGURE 18: RESPIRATORY VARIATION OF TV & MV ON 2D ECHO IN PERICARDIAL DISEASES

DETAILED STUDY OF VARIOUS PERICARDIAL DISEASES

TABLE 18. AGE AND GENDER DISTRIBUTION AMONG DIFFERENT PERICARDIAL DISEASES

		ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)		Total
Age group	< 40	6	18.2%	1	33.3%	3	25.0%	25	20.5%	35
	41-60	18	54.5%	2	66.7%	5	41.7%	43	35.2%	68
	> 60	9	27.3%	0	0.0%	4	33.3%	54	44.3%	67
SEX	F	13	39.4%	0	0.0%	3	25.0%	54	44.3%	70
	M	20	60.6%	3	100.0%	9	75.0%	68	55.7%	100
Total		33	100.0%	3	100.0%	12	100.0%	122	100.0%	170

In our study, we studied the distribution of demographical parameters among various pericardial diseases. It was seen that most of the patients diagnosed with acute pericarditis, constrictive and effusive-constrictive pericarditis were in the age group of 41-60 years. In contrast to this, patients diagnosed with pericardial effusion had highest number of patients in the age group of > 60 years.

All the four groups were comparable in their gender distribution with males having predominance as compared to females.

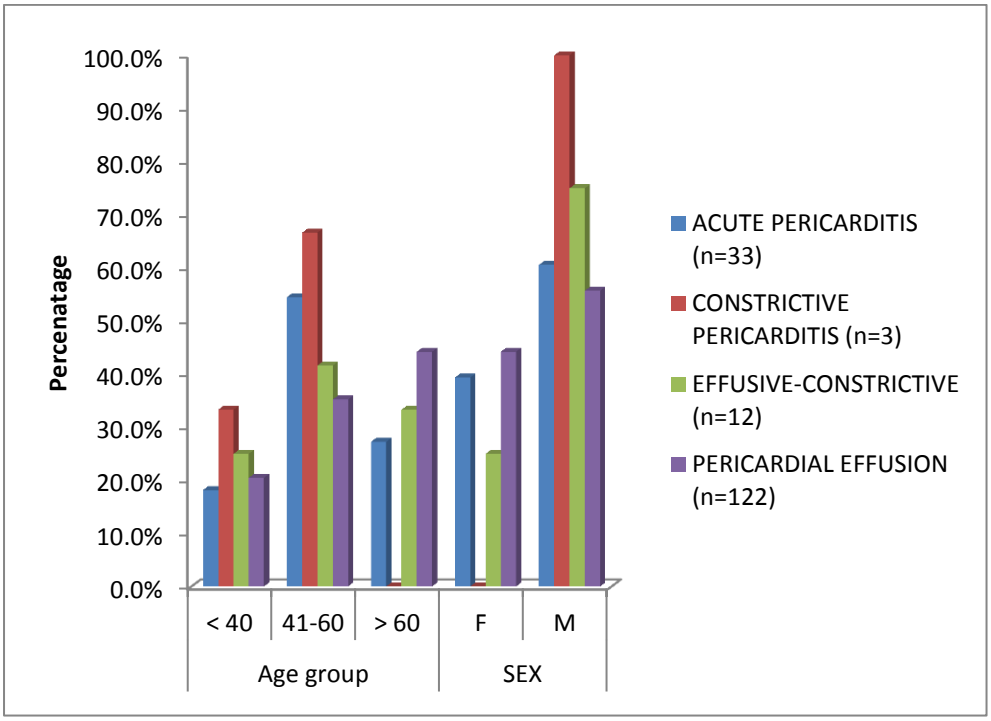

FIGURE 19. AGE AND GENDER DISTRIBUTION AMONG DIFFERENT PERICARDIAL DISEASES

TABLE 19 . OVERLAPPING SYMPTOMS IN DIFFERENT PERICARDIAL DISEASES

CHIEF COMPLAINTS	ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)		Total
SOB	15	45.45%	3	100.00%	9	75.0%	86	70.5%	113
CHEST PAIN	21	63.64%	0	0.00%	1	8.3%	17	13.9%	39
FEVER	7	21.21%	0	0.00%	6	50.0%	21	17.2%	34
COUGH	2	6.06%	1	33.33%	2	16.7%	16	13.1%	21

Pericardial diseases present with various spectrum of overlapping symptoms. In our study, among cases of acute pericarditis, chest pain (63.6%) was seen in majority of patients followed by shortness of breath (45.5%) and fever (21.2%).

All patients diagnosed with constrictive pericarditis presented with shortness of breath(100%). Patients with effusive-constrictive pericarditis had shortness of breath (75%) as the main symptom followed by fever(50%). In patients with pericardial effusion, shortness of breath was the main presenting complaint (70.49).

Cough was seen in total 21(12.3%) patients.

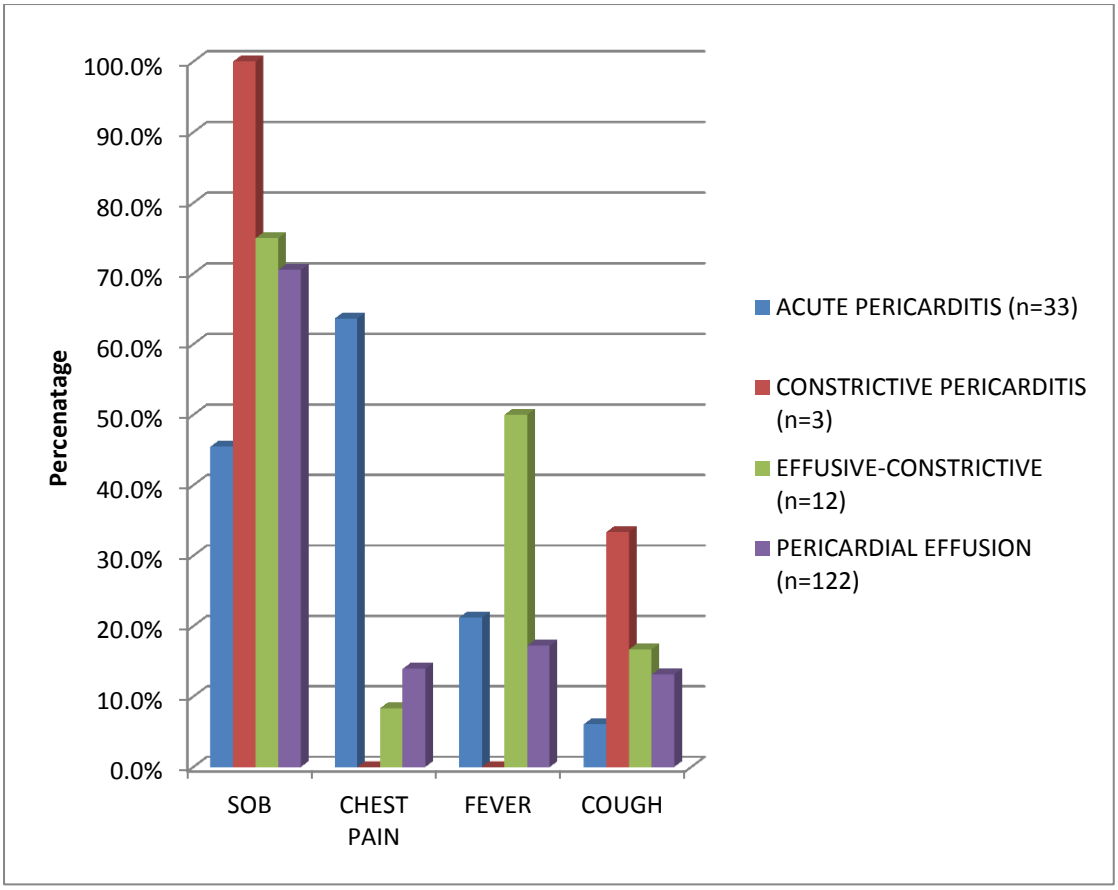

FIGURE 20. OVERLAPPING SYMPTOMS IN DIFFERENT PERICARDIAL DISEASES

TABLE 20. ETIOLOGICAL CLASSIFICATION OF VARIOUS PERICARDIAL DISEASES

BASIC DIAGNOSIS	ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)	
TUBERCULAR	1	3.03%	1	33.33%	8	66.7%	21	17.2%
IDIOPATHIC	11	33.33%	2	66.67%	0	0.0%	17	13.9%
UREMIC	8	24.24%	0	0.00%	2	16.7%	14	11.5%
post MI	9	27.27%	0	0.00%	0	0.0%	12	9.8%
HFrEF	0	0.00%	0	0.00%	0	0.0%	20	16.4%
MALIGNANCY	3	9.09%	0	0.00%	2	16.7%	13	10.7%
AUTOIMMUNE	1	3.03%	0	0.00%	0	0.0%	6	4.9%
OTHER	0	0.00%	0	0.00%	0	0.0%	6	4.9%
HYPOTHYROIDISM	0	0.00%	0	0.00%	0	0.0%	5	4.1%
HFpEF	0	0.00%	0	0.00%	0	0.0%	4	3.3%
PYOGENIC	0	0.00%	0	0.00%	0	0.0%	4	3.3%

In our study, most of the patients diagnosed with Acute pericarditis(n = 33) were found to have idiopathic (n=11, 33.33%) etiology. It was followed by post MI (n=9, 27.2%), uremic(n=8,24.2%) , malignancy (n=3,9%), autoimmune (n=1,3%) and tubercular(n=1,3%) cause in that order.

Among cases of Constrictive Pericarditis, two patients had idiopathic (66.7%) etiology and one had tubercular (33.3%) etiology.

In cases of Effusive-constrictive pericarditis most common etiology seen was tubercular (n=8, 66.7%) followed by malignancy (n=2, 16.6%) and uremic (n=2, 16.6%).

In our study, pericardial effusion was present in 71.7% (122) of total cases (cases of acute pericarditis with pericardial effusion are not included). Most common etiology of pericardial effusion seen was tubercular(17.2%) followed by heart failure with reduced ejection fraction(16.4%). Rest of the causes in decreasing order of frequency are idiopathic(13.93%), uremic(11.5%), malignancy (10.7%), post MI (9.8%), autoimmune (4.9%), tropical fever (4.9%), hypothyroidism (4.1%), heart failure with preserved ejection fraction (3.28%), and pyogenic(3.28%).

Among cases of tropical fever, dengue contributed 4% (n=5) and scrub typhus contributed 0.9% (n=1) to total cases of pericardial effusion.

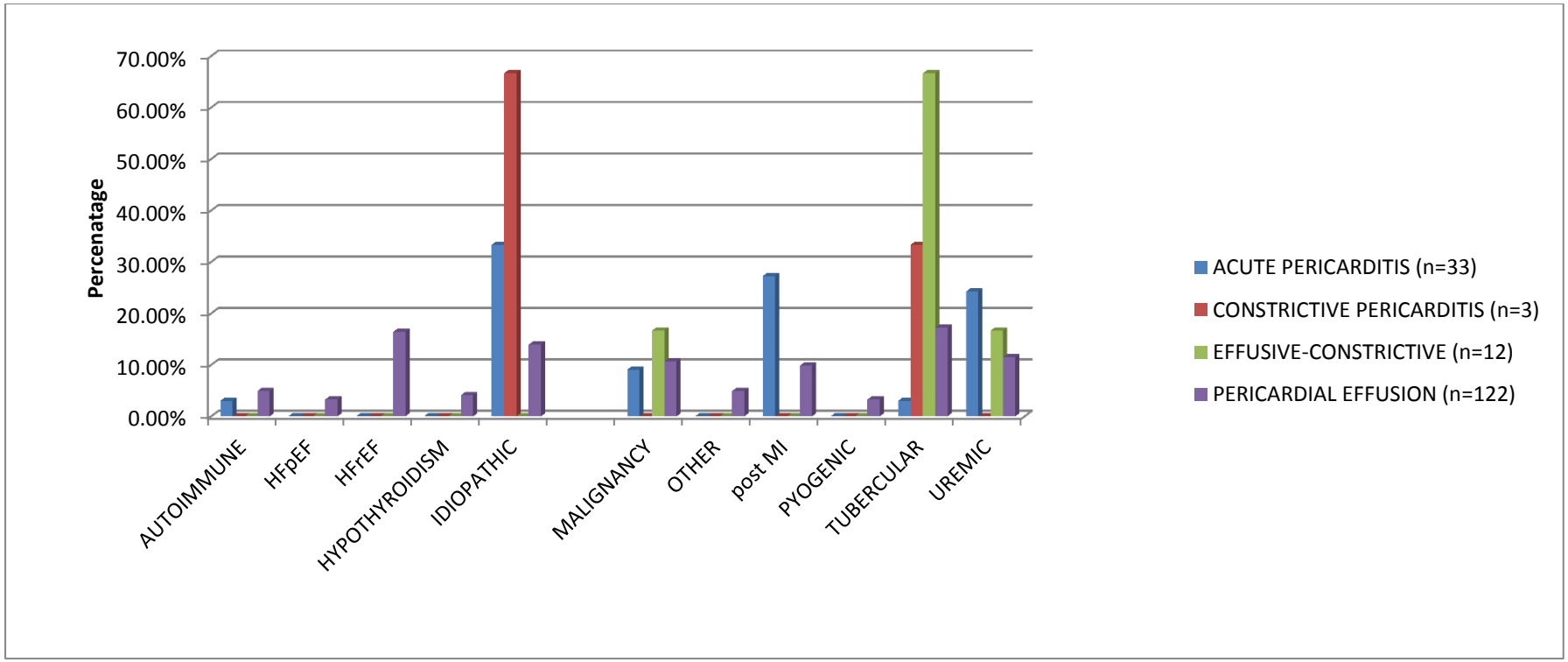

FIGURE 21 ETIOLOGICAL CLASSIFICATION OF VARIOUS PERICARDIAL DISEASES

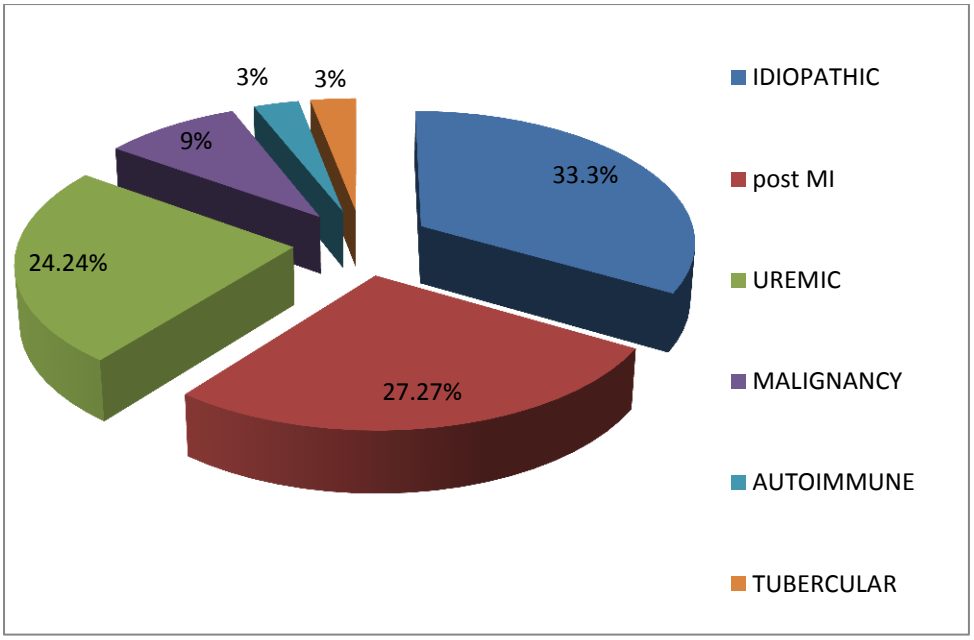

FIGURE 22: ETIOLOGIES AMONG CASES OF ACUTE PERICARDITIS

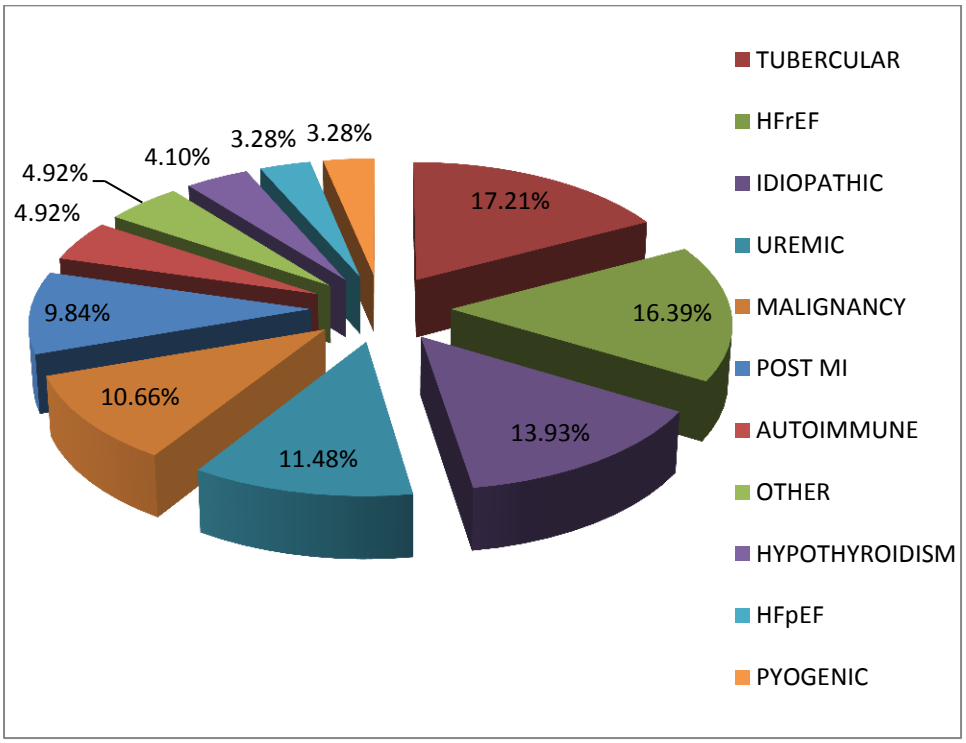

FIGURE 23: ETIOLOGIES AMONG CASES OF PERICARDIAL EFFUSION

CARDIAC TAMPONADE

TABLE 21: CARDIAC TAMPONADE IN PERICARDIAL DISEASES

CARDIAC TAMPONADE	ACUTE PERICARDITIS (n=33)		CONSTRICTIVE PERICARDITIS (n=3)		EFFUSIVE-CONSTRICTIVE (n=12)		PERICARDIAL EFFUSION (n=122)		Total
ABSENT	33	100%	3	100%	10	83.3%	104	85.2%	150
PRESENT	0	0%	0	0%	2	16.7%	18	14.8%	20

In our study, out of 170 patients with pericardial diseases, 20 patients were found to have cardiac tamponade physiology observed on ECHO. Out of 122 patients with pericardial effusion, 18 patients had tamponade physiology. 2 out of 12 patients with effusive-constrictive pericarditis had associated cardiac tamponade physiology on ECHO.

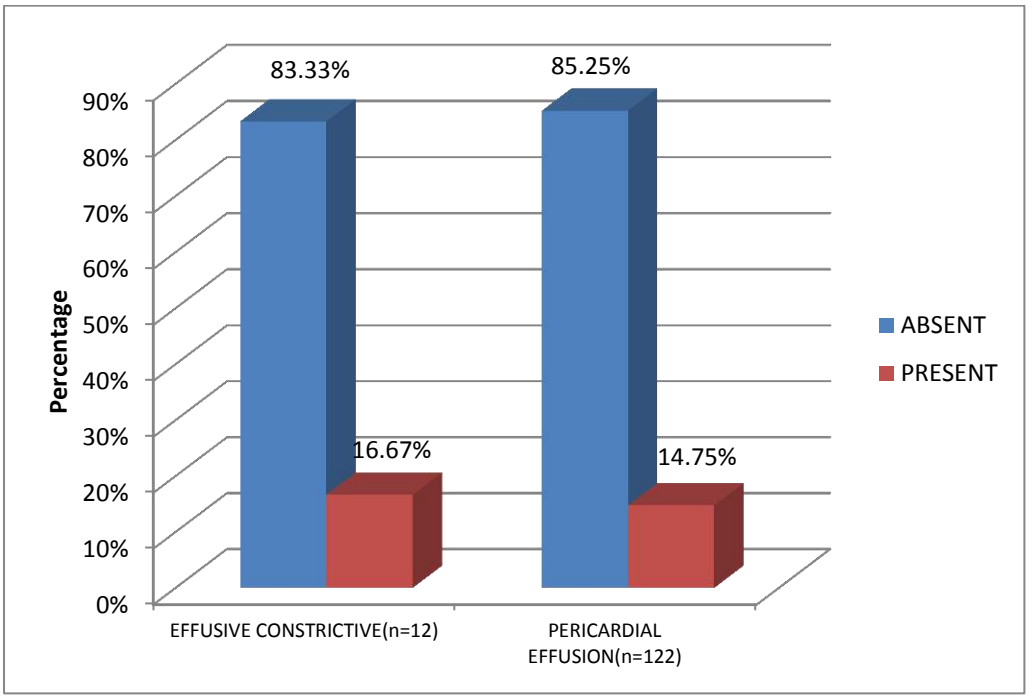

FIGURE 24 CARDIAC TAMPONADE IN DIFFERENT PERICARDIAL DISEASES

TABLE 22. CARDIAC TAMPONADE ETIOLOGY

Etiology	n	Percentage
TUBERCULAR	9	45.0%
MALIGNANCY	3	15.0%
IDIOPATHIC	2	10.0%
AUTOIMMUNE	2	10.0%
UREMIC	2	10.0%
ACS	1	5.0%
PYOGENIC	1	5.0%

All patients with cardiac tamponade underwent pericardioentesis. Most common etiology of cardiac tamponade was found to be tubercular(n=9,45%) followed by malignancy(n=3,15%), idiopathic (n=2,10%), autoimmune(n=2,10%), uremic (n=2,10%), post MI (n=1,5%) and pyogenic(n=1,5%) in decreasing order.

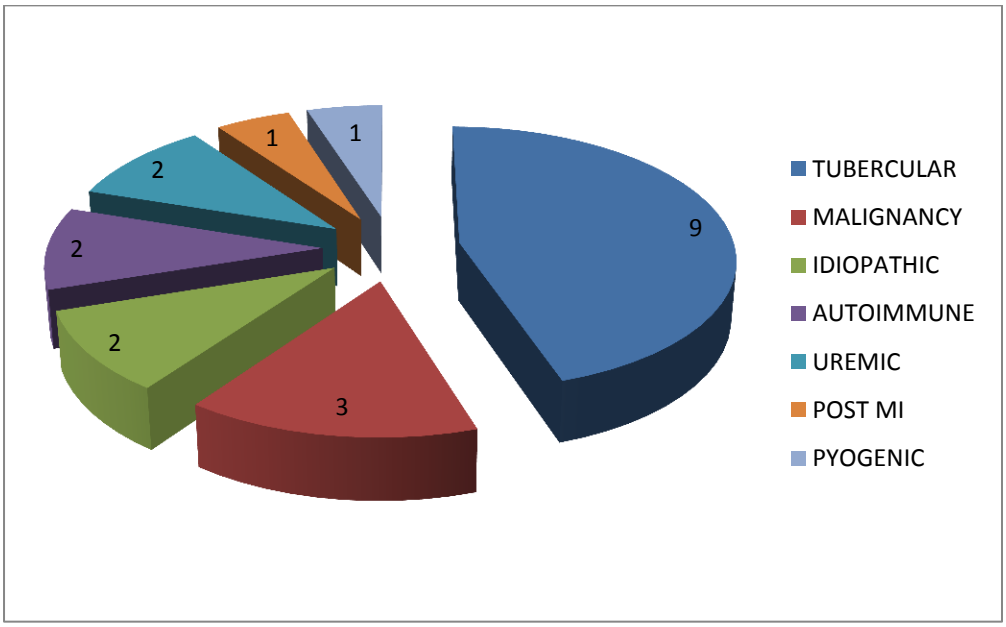

FIGURE 25. ETIOLOGY OF CARDIAC TAMPONADE

PERICARDIAL FLUID ANALYSIS

In our study, pericardial fluid analysis results were available for 32 patients. Among 32 patients, 21 patients had severe pericardial effusion and 11 patients had moderate pericardial effusion.

In 27 patients, the fluid was exudative in nature. Among 27 patients, 15 were of infectious etiology (13 tubercular and 2 pyogenic), 4 of malignant and uremic each, 3 idiopathic and 1 of autoimmune etiology.

TABLE 23: TYPE OF PERICARDIAL FLUID ASPIRATE

PERICARDIAL FLUID TYPE	No. of cases	Percentage
HEMORRHAGIC	7	21.8%
STRAW	24	75%
PURULENT	1	3.1%

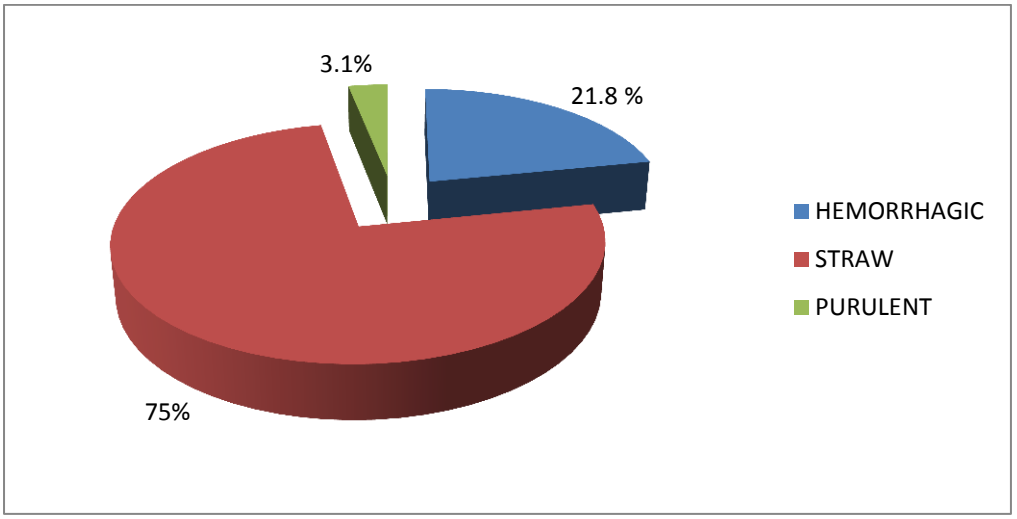

FIGURE 26. TYPE OF PERICARDIAL FLUID ASPIRATE

In our study, straw coloured aspirate was seen in majority of cases (n=24,75%). Atypical cells on malignant cytology were observed in 4(12.5%) cases. Mtb complex was detected in 2 (6.25%) cases on CBNAAT testing.

TABLE 24. ADA VALUES ON PERICARDIAL FLUID ANALYSIS

PERICARDIAL FLUID ADA	No. of cases	Percentage
< 30 (NORMAL)	9	28.13%
30-40(SUSPECT)	3	9.38%
40-60(STRONG SUSPECT)	8	25.00%
> 60(POSITIVE)	12	37.50%
Total	32	100.00%

ADA analysis of pericardial fluid came positive in 37.5% cases (n=12), 25% (n=8) cases detected as strong suspect and 9.3% (n=3) cases detected as suspect.

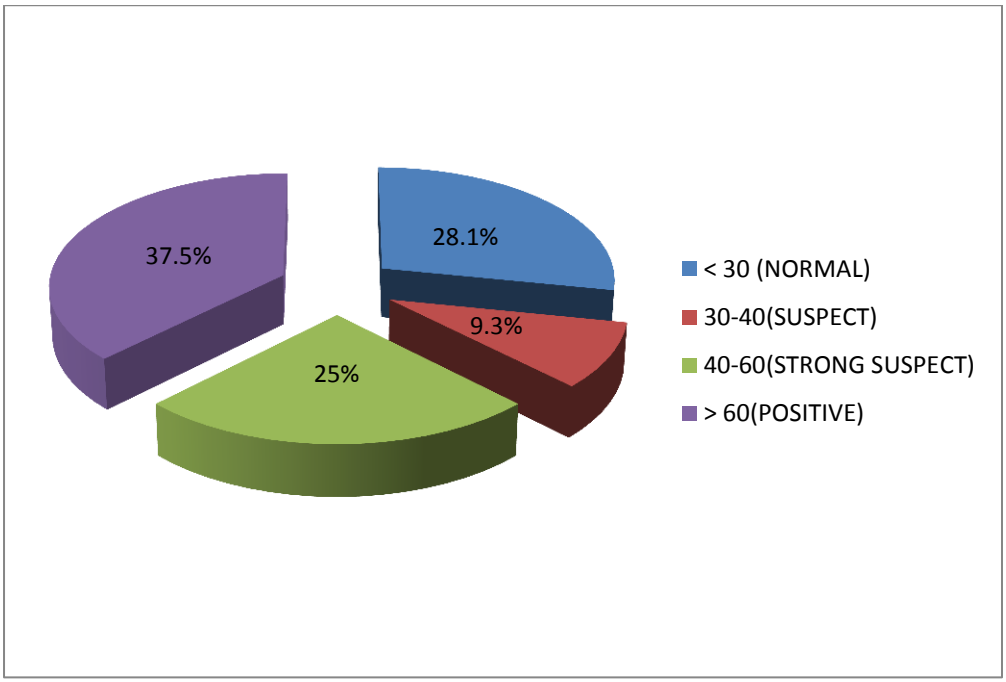

FIGURE 27. ADA ANALYSIS OF PERICARDIAL FLUID

TABLE 25. PERICARDIAL FLUID CULTURE GROWTH OBSERVED

PERICARDIAL FLUID CULTURE	No. of cases	Percentage
MRSA	1	3.1%
S. *epidermidis*	2	6.25%
S. *hemolyticus*	1	3.1%
S. *hominis*	1	3.1%
Total	5	15.6%

Pericardial fluid culture showed growth of MRSA(n=1), *S.epidermidis*(n=2), *S. hemolyticus* (n=1), and *S. hominis*(n=1). Rest of the culture reports were negative.

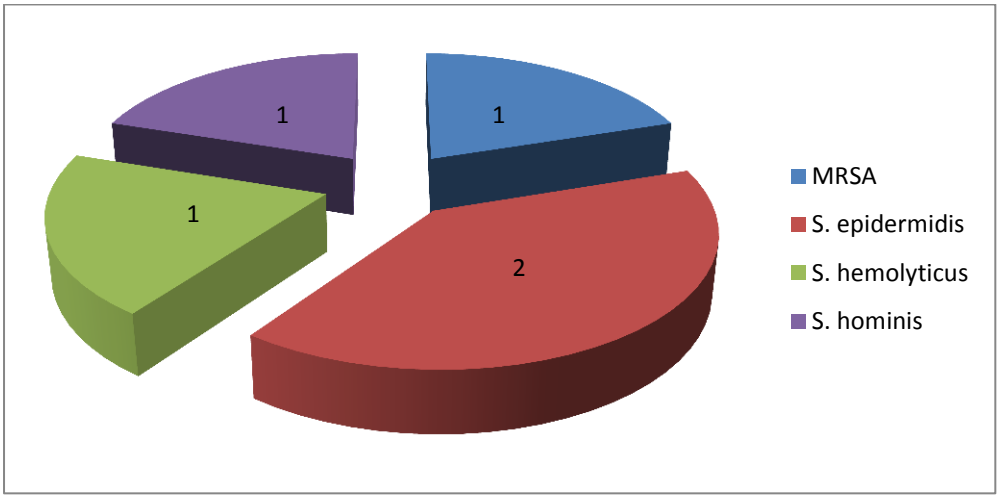

FIGURE 28. PERICARDIAL FLUID CULTURE GROWTH OBSERVED

TREATMENT MODALITIES

Table 26. MEDICAL MANAGEMENT

MEDICAL TREATMENT	No. of cases	Percentage
ASPIRIN	69	40.6%
COLCHICINE	3.0	1.8%
CORTICOSTEROIDS	46.0	27.1%
ATT	31.0	18.2%
RRT	19.0	11.2%
DIURETICS	64.0	37.6%

In our study, majority of patients were managed conservatively. Management was based on correcting the underlying pathology, giving specific drugs like aspirin/steroids and supportive management. In our study, aspirin was given in 69(40.6%), steroids were given in 46(27.1%) and colchicine was given in 3(1.8%) cases. ATT was started in 31(18.2%) cases, wherein there was established diagnosis of tuberculosis or it was added empirically. Renal replacement therapy was given in 19(11.2%) cases in patients with CKD.

TABLE 27. INTERVENTIONS DONE

TREATMENT	N	PERCENTAGE
PERICARDIOCENTESIS	28	16.5%
PLEURO-PERICARDIAL WINDOW	6	3.5%
PERICARDIECTOMY	2	1.2%

Intervention like pericardiocentesis was done in 28(16.5%) cases including 23 cases with pericardial effusion and 5 cases with effusive-constrictive pericarditis.

Surgical treatment like pleuro-pericardial window and pericardiectomy were done in 6(3.5%) cases and 2(1.2%) cases respectively.

DISCUSSION

The present study was conducted at a tertiary care hospital in North India. It is an observational study in design and it involved a detailed study of clinical profile of pericardial diseases. An attempt has been made to study the various pericardial diseases with respect to their demographic profile, clinical features and etiologies. The statistical data hence obtained was compared with previously published literature. A total of 170 patients with confirmed diagnosis of pericardial diseases were included in the study.

GENDER AND AGE WISE DISTRIBUTION

Demographic distribution in our study revealed that pericardial diseases are more common in males (58.8%) than females (41.2%). Our findings were in accordance with the previous studies conducted by Jamal Uddin et al[31] and Singh A et[32], wherein the number of males(53%) were also more than females(46%). The present study shows that the mean age of presentation in pericardial diseases was 54.7 ± 16.3 years and this number was in close range with the study conducted by Singh A et al[32], in which the mean age of the patients studied was 46.87±14.40 years. Also, in the study done by Mfeukeu-Kuate L et al[33], the mean age of the population presenting with acute pericarditis was 43 ±12 years. Similarly, according to study done by Adler Y et al[28] men aged 16–65 years were at higher risk for pericarditis (relative risk 2.02) than women in the admitted population. The age distribution in our study shows that 40% of patients in our study were in the age group of 41-60 years and 39.4% patients were above the age of 60 years.

CLINICAL PRESENTATION

Clinical manifestations in pericardial diseases vary significantly and range from asymptomatic to life-threatening presentations. In our study, the most common presenting symptoms were shortness of breath (66.5%) followed by chest pain (22.9%), and then fever (20%).

On further analysis, among cases of acute pericarditis, chest pain (63.6%) was seen in the majority of patients followed by shortness of breath(45.5%) and fever(21.2%). It was comparable to a study done by LeWinter et al[35] in which the main symptom of acute pericarditis was chest pain in more than 90% of cases, often quite severe. In contrast to this, the study done by Mfeukeu- Kuate L et al[33] revealed that the most frequent presenting complaints in acute pericarditis were cough (83.9%), precordial pain (77.4%), and dyspnea(77.4%).

Shortness of breath was the main presentation among cases of constrictive-pericarditis in our study which is similar to the study by Ling L H et al[36] where the presentation was breathlessness in 90% of cases of constrictive-pericarditis. In cases with pericardial effusion as well, shortness of breath (67.4%) was the most experienced symptom followed by chest pain (16.3%) and fever (12%). In the studies by Yaqoob et al[37] and Jamal Uddin et al[31] which included 102 and 66 cases respectively, both reported breathlessness as the most observed clinical features. Comparable to our study, breathlessness was seen in 51.4% of cases in the study done by Singh A et al[32] stating it as the commonest symptom in cases of pericardial effusion. Cough was seen in 21(12.3%) patients in our study whereas in study by Jamal Uddin[31] et al it was present in 5 (9%) patients.

PHYSICAL SIGNS

In the present analysis, the most common sign observed on physical examination was tachycardia (n=81, 47.6%) followed by raised JVP (n=70,41%). Similarly, in a study done by Jamal Uddin[31] et al which included 66 patients with pericardial effusion, the most common clinical sign observed was tachycardia (n=46;69.6%), followed by breathlessness(n=40,60%). Similar findings were seen in the study done by Yaqoob et al[37] which included 102 patients with moderate to severe pericardial effusion where tachycardia in 64%(n=65) and hypotension in 23.5%(n=24) was present. In contrast to these studies ,

in a study by Singh A et al [32] raised jugular venous pulse(55.7 %) was seen in more number of patients than tachycardia (47.1%).

Pulsus paradoxus is a typical sign of significant pericardial effusion (sensitivity of 80% and specificity of 40%[38]) .In our study, pulsus paradoxus was seen in 17 cases(10%) in contrary to 4 studies between 1991 and 1999 which gave its prevalence between 12% and 75%.[39] Also, in an Indian study done by Jamal Uddin[31] et al ,which included cases with pericardial effusion only ,pulsus paradox was found in 22(33%) patients. Similarly, in study done by yakoob et al[37] , it was seen in 36(35%) patients. Such high frequency is contributed by the fact that this study included only moderate to severe pericardial effusion cases [37], whereas in our study majority of the cases are of mild pericardial effusion.

In our case study, pericardial rub was observed in 69 % cases of acute pericarditis. LeWinter MM et al[35] reported pericardial rub in one-third of acute pericarditis cases on presentation. According to Khandaker M H et[40], audible friction rub, is present in 85% of patients with pericarditis at some time during their disease. In the study by Imazio et al[19], it is reported in 33 to 85 % of cases. Mfeukeu- Kuate L et al [33] reported in his study that pericardial rub was seen in 18(29%) patients of acute pericarditis [33].

ETIOLOGIES OF PERICARDIAL DISEASES

The most common etiology of pericardial diseases was tubercular in our study which was similar to results observed in the study done by Noubiap JJ et al [34] in which tuberculosis is the most frequent cause of pericardial diseases. According to Fowler[41] , tuberculosis is responsible for approximately 4% of cases of acute pericarditis, 7% of cases of cardiac tamponade, and, in older studies[41], 6% of instances of constrictive pericarditis.

In our study, the most common autoimmune etiology associated with pericardial illness found is SLE (2.9%) which is similar to the literature by Jiamsripong P et al[42], wherein the most common pericardial involvement among systemic inflammatory diseases is SLE. Comparable results were observed in other Indian studies done by Jamal Uddin et al and Yakoob et al which showed collagen vascular diseases as the etiology in 3.03% and 5.8% of patients [31,37].

In our study, the most common cause of acute pericarditis seen was idiopathic(n=11,33%). This finding was similar to the studies done by Mookadam F et al[2], Maisch B et al[3], and Posner[43] in which the most common etiology of acute pericarditis was viral or idiopathic. In an immunologically competent patient with classic acute pericarditis, the probability of acute idiopathic pericarditis is approximately 90% [2].

In our study, post MI pericarditis was seen in 27.2%(n=9) cases, it included both early as well late post-infarction cases. This much number of cases of post MI pericarditis were contributed by the fact that our study included cases mostly from center dedicated to cardiological diseases & interventions. This result was dissimilar to a study done by Troughton RW[44], which states that pericarditis associated with transmural myocardial infarction has become less frequent with the use of thrombolysis but still occurs in 5–10% of cases. Late post-infarction pericarditis (Dressler's syndrome) occurs in up to 5% of myocardial infarction patients and typically presents more than 1 week after infarction

Among cases with pericardial effusion, the most common etiology of effusion seen in our study was tuberculosis (17.2%) which is similar to studies done by Jamal Uddin et al[31] and Yaqoob et al[37] which also revealed tuberculosis as the leading etiological factor with the prevalence of 24.5% and 27.3% respectively. In our study, 24(14%) patients with heart failure (including both with reduced LVEF and with preserved LVEF) have been found to have pericardial effusion (mild or moderate) and it came out to be the second leading cause of

pericardial effusion. Similarly, in the study done by Kataoka H[45] ,12 out of 60 patients with chronic heart failure had small or moderate pericardial effusion. Also, according to literature by LeWinter et al[35] , patients with severe circulatory congestion may have small to moderate transudative effusions.

There was no case of pericardial effusion due to radiation injury observed in our study as the outcome & and follow up of patients were not included. In our study, 13(10.7%) patients had malignant etiology of pericardial effusion and the most common malignant etiology of pericardial effusion was lung carcinoma. Also, in the study by Yaqoob et al[37] carcinoma lung was the most common cause of pericardial effusion. A similar study conducted by Posner[43] revealed Malignant pericardial disease in 18 (58%) of 31 patients. In contrast to these studies, 114 individuals with a recent or distant history of cancer with pericardial effusions were investigated by Porte et al [46]. His results revealed the most common etiology of pericardial effusion to be of a non-malignant origin which was followed by idiopathic pericardial effusion.

Corey et al[47] investigated the etiology of pericardial effusion in 57 patients in area where TB was not prevalent. An etiologic diagnosis was made in 53 patients (93%). The most common diagnoses were malignancy (23%), viral infection (14%), radiation induced inflammation(14%), collagen vascular disease (12 %) and uremia(12%). The study by Sagrista-Sauleda et al[48] included 322 patients, 132 with moderate and 190 with severe pericardial effusion. In this series, the most common diagnosis was idiopathic-20%, neoplastic-13%, post MI-8%, uremia-6%, collagen vascular disease-5% and tubercular-2%.

In our study, only 3 cases of constrictive-pericarditis were observed due to short duration of study, two cases had idiopathic etiology and one had tubercular constrictive pericarditis. According to the study by Khandaker M H et al[40] in developed nations, idiopathic causes &

cardiac surgery are the main cause of constrictive pericarditis followed by mediastinal radiation. In developing and under-developed countries as well in immuno-compromised patients, tuberculosis is the major cause of constrictive-pericarditis but in our study tubercular etiology was only seen in one case. In a retrospective study, done by Kumar S et al[49] of constrictive-pericarditis patients who had undergone pericardiectomy from 2004 to 2019, post-operative histological diagnosis of tubercular etiology was seen in 48.2% and idiopathic in 42% of cases.

In our study, in cases of effusive-constrictive pericarditis most common etiology seen was tubercular (n=8,66.7%) followed by malignancy(n=2,16.6%) and uremic(n=2,16.6%). In a study by Noubiap JJ et al[34] patients with tuberculous pericarditis presented mostly with effusive pericarditis (79.5%) and in 15.1% and 13% of cases with effusive constrictive pericarditis and myopericarditis, respectively. In contrast to our study , the study by Syed FF et al[50] showed the most common etiologies of effusive-constrictive pericarditis as cancer, radiation injury, tuberculosis, complications following pericardiotomy, and connective-tissue disorders. Tuberculosis has been found to be the leading cause in sub-Saharan Africa likewise in India.

According to an African study[34], a large proportion of tubercular pericardial disease (up to 20%) present as cardiac tamponade. Similarly, in our study, the most common cause of cardiac tamponade was tuberculosis. Likewise, according to a study by Yaqoob et al[37] malignancy (33.3%) and tuberculosis (30%) are the leading etiologies of cardiac tamponade. It is more common in patients with a specific etiology, such as neoplastic, tuberculous, or purulent pericarditis (up to 68%)[2]. According to study done by Jiamsripong P et al[42], Cardiac tamponade may be seen in approximately 10 % of uremic pericarditis cases and 14-16% of cases with dialysis pericarditis which was comparable to numbers found in our study i.e 10% of cases with cardiac tamponade had uremic pericarditis.[42].

Total 16 (9.41%) patients were detected as COVID-19 cases, 11 were having positive SARS-COV2 antibodies and 5 were COVID RTPCR positive. Out of 5 positive COVID RTPCR patients 3 were diagnosed as idiopathic but the etiology of pericardial disease could be COVID 19 infection. Sauer F in a case series, reported three patients aged 51-84 who developed pericarditis related to COVID-19 [51] .

ECG

The pericardium is electrically silent and the ECG changes were reported in no more than 60% of cases and more common (>90%) when concomitant with myocarditis[35]. In our study, most common ECG changes found were low voltage QRS complex (n=41, 24.1%) followed by ST-segment elevation (n=37, 21.7%). PR segment depression as well as T-wave changes (flattening or inverted) seen in 3.5%(n=6) cases each and electrical alternans seen in 1.6%(n=2) cases. Similarly, in the study by Mfeukeu- Kuate L et al[33] low voltage graph was the most common finding in 54.2 % followed by elevated ST segment in 37.5%, negative T-wave in 29.2%, and lightening of PR segment in 8.3%. Unlike our study, a study done by Yaqoob et al[37] revealed the most common ECG findings to be sinus tachycardia followed by non-specific ST-T changes and low voltage QRS complexes. Although, among the least common was electrical alternans which was similar to our study.

ECHOCARDIOGRAPHY

Echocardiography is the procedure of choice for the diagnosis of pericardial effusion. The mean yearly incidence and prevalence of pericardial effusion were 3 and 9%, respectively, in a 6-year experience of the echo laboratory at Maria Vittoria hospital[35], an urban general hospital in Torino, and an Italian referral center for pericardial disorders (2000–05). Pericardial effusion is reported in about 60% of cases of acute pericarditis and is usually mild[35]. In the present study, the presence of mild pericardial effusion in acute pericarditis cases is 75%.

In the study by Singh A et al[32] and Yaqoob et al [37]echocardiography revealed severe pericardial effusion in 54(77.1%) and 37(36.3%) patients respectively whereas in our study severe pericardial effusion was seen in 25(15.1%) patients.

Associated myocardial involvement can be present in as many as 15 % of cases of pericarditis according to the study by LeWinter MM et al[35]. Fanne RA[52] concluded that among patients presenting with presumed idiopathic/viral pericarditis, myopericarditis is relatively common and has a benign evolution. In our study, it was seen in 16(48.48%) patients. Frond-like or shaggy–appearing structures in the pericardial space detected by echocardiography suggest clots, chronic inflammation, or neoplastic pericardial processes[35] and in our study, fronds were seen in 13 % of cases.

In cases of Constrictive pericarditis, most patients have a thickened pericardium. In our study, thickened pericardium is seen in 66.7 % cases of CP and 18.2 % cases of effusive-constrictive pericarditis on echocardiography. According to Posner MR et al, 18% are reported to have a normal thickness on direct histopathological examination and 28% on CT scanning[43],. In a study by Kumar S et al, pericardial thickening was seen in 94% of cases of CP who had undergone pericardiectomy[49].

As first reported by Hatle et al[53] and Oh et al[54] in a larger cohort, dynamic changes with respiration occur in patients with CP but not in patients with restrictive cardiomyopathy. Typically, patients with CP demonstrate an increase in early diastolic mitral inflow velocity of ≥25% during expiration compared with inspiration. Sensitivities and specificities of Doppler assessment of respiratory changes have been reported to be as high as 85% to 90%[55] and in our study it is observed in 100% cases of constrictive-pericarditis, 63.6% cases of effusive-constrictive pericarditis.

In our study, cardiac tamponade was seen in 11.5 % of cases detected on echocardiography. According to the study done by Mookadam F et al[2] cardiac tamponade was reported in 5-28% of cases of acute idiopathic pericarditis. In the similar studies by Singh A et al and Yakoob et al[37], 24.3% (17) patients and 38.2%(39) respectively had tamponade diagnosed through clinical and echocardiographic evaluation.

LABORATORY PARAMETERS

In our study, leukocytosis was seen in 52.4%(n=64) cases, raised ESR in 68.8%(n=84) cases, raised CRP in 23.7%(n=29) cases and positive quantitative troponin-T was seen in 18%(n=22) cases. According to a study by Little WC[1], patients with acute pericarditis usually have evidence of systemic inflammation, including leukocytosis, elevated erythrocyte sedimentation rate, and increased C-reactive protein. Troponin is frequently minimally elevated in acute pericarditis, usually in the absence of an elevated total creatine kinase. In a study by Singh A et al [32] mean ESR was 34.71±20.34 and in our study mean ESR came 55.12±31.81. In a research by Bonnefoy E et al [56], an elevation in cardiac troponin I was commonly found in patients with idiopathic acute pericarditis, notably in younger patients and those who had just been infected. Although ST-segment elevation does not always imply myocardial damage, only these patients have a large increase in cardiac troponin I.

TREATMENT

In our study intervention like pericardiocentesis was done in 21(17.2%) cases and surgical treatment like pleuro-pericardial window and pericardiectomy was done in 6(4.9%) cases and 2(1.6%) cases respectively. In the “Barcelona experience”, the overall diagnostic yield of pericardiocentesis was 19%[19]. Corey GR et al[47] analyzed the pericardial fluid and yielded a diagnosis in 15 (26%) patients, 11 of whom had malignant effusions. The examination of pericardial tissue was useful in the diagnosis of 13 (23%) patients, 8 of whom had an

infectious agent cultured. Of the 57 patients undergoing surgery, the combined diagnostic yield from both fluid and tissue was 19 patients (33%), whereas analysis in our study yielded results in 19(59%) patients, 5 of whom have culture growth observed, 2 have positive CBNAAT testing and 12 showed positive ADA values.

Analyses of the pericardial fluid specific gravity (>1015), protein level (>3.0 g/dL), fluid/serum ratio >0.5, LDH >200 mg/dL, serum/fluid >0.6, and glucose can separate exudates from transudates, but are not directly diagnostic.[16,30]In a study by Cale-Subia LN et al[57], with sample population of 44, all pericardial fluids were misclassified as exudates using the Light's criteria. Similarly, in our study, out of 32, 27 were classified as exudates which included 13 tubercular, 4 malignant, 4 uremic, 3 idiopathic, 2 pyogenic and 1 autoimmune. Meyers D G et al[58] concluded that evaluation of pericardial fluid might be limited to cell count, glucose, protein, and lactate dehydrogenase determinations plus bacterial culture and cytology.

Pericardial diseases are a significant cause of morbidity and mortality, accounting for about 4%–13% of causes of heart failure in some studies in sub-Saharan Africa[34]. More detailed epidemiologic studies are required to improve understanding of the burden of pericardial diseases. This data should be useful especially to physicians treating patients residing in regions suffering from epidemics of tuberculosis.

LIMITATIONS

1. Small sample size, single-centered study.
2. Patients less than 18 years of age were not taken in this study.
3. HsCRP was not included in this study.
4.Follow-up of patients was not included in our study so recurrent pericarditis cases were not detected.

SUMMARY AND CONCLUSION

SUMMARY

1. The mean age of presentation was 54.7 ± 16.3 years and there were 100 males(58.8%) and 70 females(41.2%) out of 170 patients in our study.
2. Shortness of breath was the most commonly presented symptom followed by chest pain and fever.
3. The most common sign observed on physical examination was tachycardia followed by raised JVP. In 69.6 % of cases of acute pericarditis pericardial rub was noticed.
4. Specific ECHO findings reported in our study were exudative strands or fronds in 12.9%, cardiac tamponade in 11.8% and effusive-constrictive physiology in 7.1% of cases.
5. Spectrum of pericardial diseases observed in our study is pericardial effusion in 71.8%, acute pericarditis in 19.4%, effusive-constrictive-pericarditis in 7.1%, and constrictive pericarditis in 1.8% of cases.
6. Comorbidities like CKD was present in 20%, hypothyroidism in 11.2 %, connective tissue disorder in 4.7%, COPD in 3.5%, and malignancy in 2.9% of cases in our study.
7. The most common etiology of pericardial disease came out to be tubercular(18.2%). Other etiologies like uremic pericarditis was found in 14.1%, malignancy in 10.6% , and autoimmune in 4.1% of cases.
8. Cardiac tamponade is observed in 20(11.8%) patients and pulsus paradox was seen in 17(10%) cases. Tuberculosis (n=9,45%) is the most common etiology of tamponade in our study followed by malignancy(n=3,15%) and uremic pericarditis (n=2,10%).
9. In our study, 27 out of 32 were classified as exudates which included 13 tubercular, 4 malignant, 4 uremic, 3 idiopathic, 2 pyogenic and 1 autoimmune.

10. Pericardial fluid analysis in our study yielded results in 19(59%) patients, 5 of whom have culture growth, 2 have positive CBNAAT testing and 12 showed strongly positive ADA values.

CONCLUSION

Our study demonstrated that tuberculosis is the most frequent etiology of pericardial disease followed by idiopathic and uremic. The higher frequency of tuberculosis and uremic pericarditis probably reflects the spectrum of diseases prevailing in our area, where tuberculosis is still common and end-stage renal disease is frequent. The high prevalence of tuberculosis in India is attributable to poor socio-economic status and overcrowding. The most common cause of acute pericarditis in our study remained idiopathic similar to western countries, which could be due to the non-detection of viral pathologies. Tuberculosis also came out to be the leading etiology of cardiac tamponade and thus mandating its early diagnosis. This study would help in early diagnosis and prompt treatment of patients with pericardial effusion especially in remote areas which remains a challenging problem for diagnosis and treatment.

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| | | | | | | | CHIEF COMPLAINTS | | | | MEDICAL CO-MORBIDITIES | | | | | | |

SR NO	MRD NO	PERICARDIAL FLUID ANALYSIS (IF INDICATED)																		CECT/ MRI FINDINGS	BASIC DIAGNOSIS					MEDICAL TREATMENT									PERICARDIOCENTESIS	SURGICAL TREATMENT		COMMENT	COVID
		LVEF	LVDD TYPE	OTHER	TYPE (HEMORRHAGIC/ STRAW COLORED/ CHYLOUS/ OTHER)	SMEAR FOR AFB PLEURAL FLUID	MALIGNANT CYTOLOGY	CYTOLOGY			BIOCHEMISTRY				FLUID CULTURE	FLUID PH	CB-NAAT	LDH	OTHERS			MALIGNANCY	AUTOIMMUNE	OTHER	ASPIRIN	COLCHICINE	IBUPROFEN	CORTICOSTEROIDS	ATT	RRT	DIURETICS	OTHER		PLEURO-PERICARDIAL WINDOW	PERICARDIECTOMY		
								TLC	LYMPHOCYTES	NEUTROPHILS	GLUCOSE	PROTEIN	FLUID PRO/ S.PRO	ADA																							
1	875402	60																			TUBERCULAR	PERICARDIAL EFFUSION				0.5 MG BD		MEDROL 32MG	Y								
2	878896	55																		Y	UREMIC	PERICARDIAL EFFUSION								Y							
3	878921	60			STRAW	N	N	200	90	10	96	4.5	0.66	25	N	7		1009			IDIOPATHIC	PERICARDIAL EFFUSION										Y					
4	879321	42		RWMA																Y	IDIOPATHIC	PERICARDIAL EFFUSION															
5	895534	55			HEMORRHAGIC	N	N	1000	90	10	143	4.2	0.76	9.28	N	7.1					MALIGNANCY	PERICARDIAL EFFUSION	NEUROENDOCRINE TUMOUR										Y		EXPIRED	
6	645125	60	I																	Y	TUBERCULAR	PERICARDIAL EFFUSION				75			WYSOLONE 40MG	Y						
7	903352	60																			MALIGNANCY	PERICARDIAL EFFUSION	SCC LUNG											CHEMOTHERAPY		LOCALISED	
8	882679	30	III																		UREMIC	PERICARDIAL EFFUSION									Y						
9	918086	60																			TUBERCULAR	PERICARDIAL EFFUSION								Y						PLEURAL TB	
10	972609	60																			OTHER	PERICARDIAL EFFUSION			DENGUE FEVER						Y						
11	972054	60																			MALIGNANCY	PERICARDIAL EFFUSION	ADENOCA LUNG													DAMA	
12	924067	60																			MALIGNANCY	PERICARDIAL EFFUSION	CA BREAST														
13	923108	60																		Y	AUTOIMMUNE	PERICARDIAL EFFUSION		SJOGRENS					EFFCORLIN								
14	920811	60			STRAW		N				47	5.7	0.95	144	STAPH EPIDERMIDIS	7.4	N	1160			IDIOPATHIC	PERICARDIAL EFFUSION										Y					
15	922932	28	III																		IDIOPATHIC	PERICARDIAL EFFUSION									Y						
16	943032	60			STRAW	N	N	500	90	10	103	2.1	0.31	12		7.4	N	77		Y	TUBERCULAR	EFFUSIVE-CONSTRICTIVE							WYSOLONE 30MG	Y		Y		Y		
17	925040	24																			HFrEF	PERICARDIAL EFFUSION										Y					
18	925282	42			HEMORRHAGIC	N	N	5400	30	70	112	7.4	1.06	34	N	7.5	N	488		Y	IDIOPATHIC	PERICARDIAL EFFUSION				75								Y					
19	926903	60																			MALIGNANCY	PERICARDIAL EFFUSION	PAPILLARY SEROUS CARCINOMA														
20	817714	28		GHK OF LV &RV																Y	UREMIC	PERICARDIAL EFFUSION									Y						
21	811785	40																			MALIGNANCY	ACUTE PERICARDITIS	METASTATIC ADENOCARCINOMA LUNG											INDOCAP				
22	930483	60		ANNULUS PARADOX	HEMORRHAGIC	N	N	400	70	30	52	5.6	0.86	153	N	7.5		923			TUBERCULAR	EFFUSIVE-CONSTRICTIVE									Y			Y		ANNULUS PARADOXUS	
23	929305	60			STRAW	N	ATYPICAL	1500	90	10	47	5	0.81	412	N	7.5		4773		Y	MALIGNANCY	EFFUSIVE-CONSTRICTIVE	NHL						DEXONA					Y			BIOPSY-T CELL LYMPHOMA	
24	924588	28		GHK OF LV																	UREMIC	PERICARDIAL EFFUSION															
25	936054	60																			IDIOPATHIC	PERICARDIAL EFFUSION			INFECTIVE ENDOCARDITIS	75					Y						
26	931244	30		GHK OF LV	STRAW	N	N	400	90	10	132	4.4	0.83	32	N	7.5	N	656			UREMIC	PERICARDIAL EFFUSION											Y				
27	930264	30	III	RWMA	STRAW	N	N	1800	90	10	143	5.2	0.68	41	N	7.6	Y	314			TUBERCULAR	PERICARDIAL EFFUSION							WYSOLONE	Y				Y			
28	185353	60																			HFpEF	PERICARDIAL EFFUSION															
29	939961	35		GHK OF LV &RV																	IDIOPATHIC	PERICARDIAL EFFUSION														DAMA	
30	938322	42																			IDIOPATHIC	PERICARDIAL EFFUSION			INFECTIVE ENDOCARDITIS												
31	939141	20	III	DCMP																	HFrEF	PERICARDIAL EFFUSION			DCMP	75					Y						
32	846381	60																			IDIOPATHIC	PERICARDIAL EFFUSION			RA,HCM				MEDROL								
33	850791	28		GHK OF LV &RV																	AUTOIMMUNE	ACUTE PERICARDITIS		RA/SLE OVERLAP SYNDROME					WYSOLONE			Y					
34	939927	35		RWMA																	post MI	PERICARDIAL EFFUSION				150						Y	DIGOXIN				
35	940521	20	III	DCMP																	HFrEF	PERICARDIAL EFFUSION				75						Y					
36	921413	35		GHK OF LV																	UREMIC	PERICARDIAL EFFUSION									Y	Y					
37	938273	30		RWMA																Y	post MI	PERICARDIAL EFFUSION				75							DIGOXIN				
38	936592	60																			IDIOPATHIC	PERICARDIAL EFFUSION															
39	513345	40		RWMA																	post MI	PERICARDIAL EFFUSION				75											
40	248337	26	II	RWMA																	UREMIC	ACUTE PERICARDITIS				75											
41	848687	20	II	RWMA																	post MI	PERICARDIAL EFFUSION			AWMI	75											
42	962467	32	II	RWMA																	post MI	ACUTE PERICARDITIS				75											
43	981474	60																			UREMIC	PERICARDIAL EFFUSION									Y						
44	979506	46		GHK OF LV																Y	TUBERCULAR	PERICARDIAL EFFUSION								Y						MANTOUX +	

| | | | | | | | CHIEF COMPLAINTS | | | | MEDICAL CO-MORBIDITIES | | | | | | |

SR NO	MRD NO	PERICARDIAL FLUID ANALYSIS (IF INDICATED)																		CECT/ MRI FINDINGS	BASIC DIAGNOSIS					MEDICAL TREATMENT								PERICARDIOCENTESIS	SURGICAL TREATMENT		COMMENT	COVID
		LVEF	LVDD TYPE	OTHER	TYPE (HEMORRHAGIC/ STRAW COLORED/ CHYLOUS/ OTHER)	SMEAR FOR AFB PLEURAL FLUID	MALIGNANT CYTOLOGY	CYTOLOGY			BIOCHEMISTRY				FLUID CULTURE	FLUID PH	CB-NAAT	LDH	OTHERS			MALIGNANCY	AUTOIMMUNE	OTHER	ASPIRIN	COLCHICINE	IBUPROFEN	CORTICOSTEROIDS	ATT	RRT	DIURETICS	OTHER		PLEURO-PERICARDIAL WINDOW	PERICARDIECTOMY		
								TLC	LYMPHOCYTES	NEUTROPHILS	GLUCOSE	PROTEIN	FLUID PRO/ S.PRO	ADA																						
45	982014	60																		Y	MALIGNANCY	PERICARDIAL EFFUSION	CA LUNG											CHEMOTHERAPY				EXPIRED	
46	986830	60			PURULENT	N	N	1800	20	80	113	2.6	0.44	93	MRSA	7	N	4327			PYOGENIC	PERICARDIAL EFFUSION													Y	Y		FIBRINOUS PERICARDITIS	
47	997844	60																			UREMIC	PERICARDIAL EFFUSION										Y	Y					DAMA	
48	986259	36		GHK OF LV	STRAW	N	N	1800	10	90	5	4.3	0.63	45	STAPHHOMINIS	7.5		1416		Y	AUTOIMMUNE	PERICARDIAL EFFUSION		SLE					WYSOLONE				HCQs	Y				
49	759302	55																			AUTOIMMUNE	PERICARDIAL EFFUSION		SLE					WYSOLONE			Y	AMIODARONE					
50	874615	36	II	RWMA																	IDIOPATHIC	PERICARDIAL EFFUSION				150						Y					LOCALISED	
51	1003587	35	II	RWMA																	post MI	PERICARDIAL EFFUSION			AWMI	150												
52	882698	55																			IDIOPATHIC	PERICARDIAL EFFUSION												Y				
53	140417	60																			UREMIC	PERICARDIAL EFFUSION							MEDROL 32MG			Y						
54	898013	34	II	DCMP																	HFrEF	PERICARDIAL EFFUSION										Y						
55	1003144	60			STRAW	N	N	500	95	5	181	5	0.66	43	N			188		Y	TUBERCULAR	PERICARDIAL EFFUSION								Y			NAPROXEN	Y				ab+
56	1001495	60																		Y	IDIOPATHIC	PERICARDIAL EFFUSION			AORTIC ULCER						Y							
57	1003288	32		GHK OF LV																	IDIOPATHIC	PERICARDIAL EFFUSION				75						Y						
58	338405	60																			OTHER	PERICARDIAL EFFUSION			DENGUE FEVER													
59	983780	55			STRAW	N	N	1200	90	10	218	4.2	0.71	11		7.5		128			UREMIC	PERICARDIAL EFFUSION										Y		Y				
60	987731	55																		Y	TUBERCULAR	EFFUSIVE CONSTRICTIVE				75				Y							PLEURAL ADA+	
61	1023242	60																		Y	AUTOIMMUNE	PERICARDIAL EFFUSION		SLE WITH LUPUS NEPHRITIS								Y	RITUXIMAB					
62	927448	60																		Y	MALIGNANCY	PERICARDIAL EFFUSION	ADENOCA LUNG										CHEMOTHERAPY				LOCALISED	
63	626258	60																		Y	TUBERCULAR	PERICARDIAL EFFUSION				75				Y								
64	173156	42		RWMA																	HFrEF	PERICARDIAL EFFUSION				75						Y						
65	1001295	60																			OTHER	PERICARDIAL EFFUSION			SCRUB TYPHUS							Y					AORTIC ULCER ?INTRAMURAL HEMATOMA	
66	1061675	22	III	DCMP																	HFrEF	PERICARDIAL EFFUSION			RHD	75						Y						
67	1034104	42																		Y	TUBERCULAR	EFFUSIVE-CONSTRICTIVE							WYSOLONE 40MG	Y								
68	1062834	60																		Y	TUBERCULAR	EFFUSIVE-CONSTRICTIVE							WYSOLONE 40MG	Y								ab+
69	809504	22	IV	DCMP																	HFrEF	PERICARDIAL EFFUSION				75						Y						
70	1058020	32		RWMA																	post MI	PERICARDIAL EFFUSION			AWMI	75						Y						ab+
71	1064227	60			STRAW	N	N	1500	60	40	82	5.9	0.79	66	N	7.4	N	1555			TUBERCULAR	PERICARDIAL EFFUSION								Y						Y	GRANULOMATOUS LYMPHADENITIS/ FIBRINOUS PERICARDITIS	
72	1063605	60																			TUBERCULAR	CONSTRICTIVE PERICARDITIS								Y							DAMA	
73	31298	45		GHK OF LV																	UREMIC	PERICARDIAL EFFUSION															DAMA	rtpcr+
74	1067842	55																			IDIOPATHIC	PERICARDIAL EFFUSION				75												
75	1091721	44		GHK OF LV																	IDIOPATHIC	ACUTE PERICARDITIS																
76	1102305	32		RWMA																Y	UREMIC	ACUTE PERICARDITIS																rtpcr+
77	1106940	60																			IDIOPATHIC	ACUTE PERICARDITIS																rtpcr+
78	458919	60																			UREMIC	PERICARDIAL EFFUSION										Y					PULMONARY ASPERGILLOSIS/LOCALISED	
79	211512	28	II	RWMA																	HFrEF	PERICARDIAL EFFUSION				150												
80	492838	45		DCMP																Y	TUBERCULAR	EFFUSIVE CONSTRICTIVE				75												ab+
81	1018511	26		RWMA																	post MI	PERICARDIAL EFFUSION				150						Y						
82	908517	60			STRAW	N	N	1000	35	65	120	2.8	0.55	26	N	7.5	N	127		Y	TUBERCULAR	PERICARDIAL EFFUSION								Y				Y				
83	970666	60																			OTHER	PERICARDIAL EFFUSION			DENGUE FEVER													
84	998388	35		GHK OF LV	STRAW	N	N	300	35	65	101	3.9	7.8	50	N	7.2	N	156			TUBERCULAR	EFFUSIVE CONSTRICTIVE							MEDROL 4MG	Y						Y	PERICARDIAL TB ON BIOPSY	
85	1125724	46		GHK OF LV																Y	MALIGNANCY	EFFUSIVE CONSTRICTIVE	AML										CHEMOTHERAPY					ab+
86	115045	60		ANNULUS PARADOX																	IDIOPATHIC	CONSTRICTIVE PERICARDITIS			CAD	75											ANNULUS PARADOXUS	
87	977722	60			STRAW	N	ATYPICAL CELLS							42			N			Y	TUBERCULAR	EFFUSIVE CONSTRICTIVE							MEDROL 16MG	Y				Y			EXPIRED	
88	1069918	60			STRAW	N	N	800	95	5	42	5.2	1.13	78	N	7.2	N	471		Y	TUBERCULAR	PERICARDIAL EFFUSION								Y				Y				
89	154387	18		GHK OF LV																	HFrEF	PERICARDIAL EFFUSION				75												
90	498552	28	III	RWMA																	post MI	PERICARDIAL EFFUSION				75						Y						

							CHIEF COMPLAINTS				MEDICAL CO-MORBIDITIES																CLINICAL EXAMINATION									CHEST X-RAY		LAB FINDINGS																ECG FINDINGS								2D ECHO FINDINGS						
SR NO	MRD NO	NAME	AGE	SEX	DOA	DOD	SOB	CHEST PAIN	FEVER	OTHER	DM	HTN	IHD	TB	MALIGNANCY	AUTOIMMUNE	CKD	COPD	HYPOTHYROIDISM	OTHER	H/O CARDIAC SURGERY	PREVIOUS H/O PERICARDIAL DISEASE	BP	HR	SP02	RR	JVP	LAD	PERICARDIAL RUB	PERICARDIAL KNOCK	PULSUS PARADOXUS	CARDIOMEGALY	PLEURAL EFFUSION	HB	TLC	PLATELETS	ESR	CRP	B.UREA	CREATININE	TOTAL PROTEINS	ALBUMIN	BNP	OTHERS	ST SEGMENT CHANGES	PR SEGMENT CHANGES	ELECTRICAL ALTERNANS	T WAVE LOW/ FLATTENED/ INVERTED	LOW VOLTAGE ECG	OTHERS	MILD/ MODERATE/ SEVERE PERICARDIAL EFFUSION	THICKENED PERICARDIUM	EXUDATIVE STRANDS/FRONDS	EFFUSIVE-CONSTRICTIVE	CARDIAC TAMPONADE	IVC COLLAPSE/ NORMAL/ DISTENDED	RESPIRATORY VARIATION OF MV & TV VELOCITY
91	1106186	AV	67	M	10.5.21	17.5.21	N	N	N	VOMITING	N	N	N	N	N	N				PLEURAL TB	N	N	130/80	117	98	18	R	N	N	N	N	N	N	9.1	9.1	614	105	117	66	2.1	7	2.8													MILD						
92	1110	VK	55	M	18.5.21	24.5.21	Y	N	N	N	Y	Y	N	N	N	N				N	N	N	130/90	108	90	23	R	N	N	N	N	N	N	12	9.2	223	33	71	56	1.3	6.1	3.6	1150								MOD						
93	1109550	SK	27	F	17.5.21	19.5.21	Y	N	N	N	N	N	N	N	N	N			Y	N	N	N	120/70	76	95	19	N	N	N	N	N	N	N	8.7	14	136	60		27	0.5	5.4	2.7									MILD					C	
94	67727	JS	60	M	11.5.21	18.5.21	N	N	N	COUGH	N	Y	N	N	N	N				RA	N	N	110/70	115	97	18	N	N	Y	N	N	Y	N	10	20	449	120	136	32	1	6.4	3.5	131								AF	MOD		Y				
95	1112169	B	24	F	22.5.21	8.6.21	Y	N	N	N	N	N	N	N	N	N				RHD	N	N	110/70	80	94	17	N	N	N	N	N	Y	N	9.6	13	305	100		28	0.4	6	3.31	5								AF	SEVERE		Y				
96	1025978	RK	70	M	10.2.21	17.2.21	N	Y	N	N	N	Y	N	N	N	N				N	N	N	110/70	62	94	20	N	N	N	N	N	N	N	13	13	241	54		24	0.7	5.9	0.7	206		Y													
97	1028083	DK	97	M	14.2.21	22.2.21	N	N	N	SYNCOPE	Y	N	N	N	N	N	Y			N	N	N	120/80	110	96	18	N	N	N	N	N	N	N	8.4	8.7	142	100		61	2.6	6.3	3.1		TROP-T +							MILD						
98	64818	HS	71	M	15.2.21	18.2.21	N	N	N	GBW	N	Y	N	N	N	N				RHD	N	N	110/70	142	94	20	N	N	N	N	N	Y	N	14	8.2	144			45	1.2	6.8	3.5								AF	MILD						
99	1054167	BS	70	M	16.3.21	30.3.21	Y	N	N	N	Y	Y	N	N	N	N					N	N	110/70	104	89	22	R	N	N	N	N	N	N	13	11	153	56		35	1.1	6.9	3.62									MILD						
100	1035436	NT	49	M	26.2.21	18.3.21	N	Y	N	N	N	N	N	N	N	N				N	N	N	110/70	80	93	18	N	N	N	N	N	N	N	14	9.6	271	75		26	0.9	7.4	4.1	5		Y							MOD						
101	517854	JN	76	M	17.3.21	24.3.21	N	Y	N	N	Y	N	N	N	N	N	Y			AV DISEASE	N	N	110/70	80	94	18	N	N	N	N	N	Y	N	10	13	264	92		83	2.1	7	3.3	380	TROP-T +	Y						RBBB	MOD						
102	1124199	AK	65	M	21.6.21	25.6.21	Y	N	N	ANASARCA	N	Y	N	N	N	N		Y		COR-PULMONALE	N	N	120/90	136	85	25	R	N	N	N	N	Y	N	16	8.9	186			81	2.2	4.8	2.55	1550									MILD						
103	1117128	CS	64	M	3.6.21	16.6.21	N	N	N	PALPITATIONS	Y	Y	N	N	N	N				N	N	N	120/70	38	89	22	R	N	N	N	N	Y	N	14	12	140	35		64	1.3	6.7	3.6	484	TROP-T +	Y						CHB	SEVERE			Y			
104	1123900	HS	65	M	17.6.21	23.6.21	Y	N	N	N	Y	Y	N	N	N	N				N	N	N	130/80	70	82	22	R	N	N	N	N	Y	N	14	9	256			46	0.9	6.5	3.5	1180	TROP-T +							LBBB	MILD						
105	1121836	BS	60	M	12.6.21	18.6.21	N	Y	N	N	N	N	N	N	N	N				N	N	N	120/70	80	92	20	N	N	N	N	N	Y	N	14	11	190	15		25	0.9	6.4	4.43										MILD						
106	1127677	SC	58	M	25.6.21	30.6.21	N	Y	N	N	N	Y	N	N	N	N				N	N	N	110/70	78	96	19	N	N	N	N	N	Y	N	16	16	248	13		37	1.2	6.5	3.4	394	TROP-T +								MILD						
107	1026573	CL	61	M	11.2.21	21.2.21	N	N	N	EPIGASTRIC PAIN	N	N	N	N	N	N				N	N	N	110/70	76	98	16	N	N	N	N	N	N	N	13	21	224	52	127	65	1.6	6.4	3.7	1470	TROP-T +	Y							MILD						
108	1000252	HR	59	M	15.2.21	19.2.21	N	Y	N	N	Y	Y	N	N	N	N				N	N	N	130/70	68	95	17	N	N	N	N	N	N	N	14	13	172			30	0.6	6.1	3.5			Y							MILD						
109	998399	SG	78	F	15.12.20	17.12.20	Y	N	N	N	Y	Y	N	N	N	N			Y	N	N	N	150/80	74	95	19	N	N	N	N	N	N	N	9.3	12	212	35		15	0.6	6.1	3										MILD						
110	964910	GJ	43	M	12.9.20	18.9.20	N	Y	N	N	N	N	N	N	N	N				N	N	N	140/80	86	97	20	N	N	N	N	N	N	N	14	8.3	225	20		16	0.7	7.5	4.39	128		Y							MOD						
111	1128213	PP	53	F	26.6.21	30.6.21	N	N	N	PALPITATIONS	N	N	N	N	N	N			Y	N	N	N	80/P	56	92	20	N	N	N	N	N	N	N	13	17	244			51	1.2	7	4.1	1650	TROP-T +				Y				MILD						
112	1124360	MP	72	M	18.6.21	30.6.21	N	N	N	DROWSINESS	N	Y	Y	N	N	N				N	N	N	170/100	100	96	17	N	N	N	N	N	N	N	13	10	275			17	1.1	8.2	4.1	67									MILD						
113	1115045	RC	59	M	24.6.21	30.6.21	Y	N	N	EPIGASTRIC PAIN	Y	N	N	N	N	N				N	N	N	100/60	128	88	20	R	N	N	Y	N	N	N	8.9	12	412	100	222	29	0.8	7.1	3.55	61									MILD	Y					Y
114	763166	SK	78	F	21.6.21	28.6.21	Y	N	N	N	N	N	N	N	N	N				N	N	N	110/60	106	82	22	R	N	N	N	N	Y	Y	12	13	61	10		38	1.4	5.9	3.38	5000			Y						MOD					D	
115	634648	SN	62	F	18.6.21	26.6.21	N	N	N	PEDAL EDEMA	Y	Y	N	N	N	N		Y		N	N	N	110/80	58	96	17	N	N	N	N	N	N	N	10	8	310	100		13	0.8	6.7	3.7										MILD						
116	1121064	GS	23	M	11.6.21	25.6.21	Y	N	N	COUGH	N	N	N	N	N	N				N	N	N	100/60	138	90	24	R	Y	N	N	Y	Y	Y	13	10	240	20		26	0.8	6.5	4.17							Y			SEVERE				Y		
117	1131552	YS	42	M	3.5.21	14.5.21	N	Y	Y	COUGH	N	N	N	N	N	N				N	N	N	110/70	110	94	20	R	N	N	N	Y	Y	N	13	11	212			24	0.6	6.9	3.2							Y			SEVERE				Y		
118	980469	NR	27	F	27.10.20	1.11.20	Y	Y	N	COUGH	N	N	N	Y	N	N				N	N	N	100/60	110	95	22	N	Y	Y	N	N	N	N	11	10	277	111		32	0.2	7.4	2.98			Y							MILD						
119	1029222	MK	64	F	8.5.21	21.5.21	Y	N	N	N	N	Y	N	N	N	N	Y		Y	N	N	N	120/80	80	91	19	N	N	N	N	N	Y	Y	7.2	5.6	114			67	5.6	7.2								Y			SEVERE			Y			
120	1128226	MK	67	M	26.6.21	4.7.21	Y	N	Y	N	N	N	N	N	N	N				N	N	N	100/60	84	89	24	R	N	N	N	N	Y	N	9.5	16	307	89		186	3.3	7.3	4							Y	AF	MOD	Y					
121	1105479	MS	65	M	8.5.21	21.5.21	Y	N	N	N	N	Y	N	N	N	N			Y	N	N	N	120/80	114	92	19	N	N	N	N	N	Y	N	11	6	218	40		18	0.5	7.5	3.7	30						Y			SEVERE		Y				
122	1031343	KK	65	F	19.2.21	25.2.21	Y	N	N	N	Y	Y	N	N	Y	N			Y	N	N	N	80/60	120	79	28	R	N	N	N	N	Y	N	12	16	158			34	0.5	5.9	3.4							Y	RBBB	SEVERE				Y		
123	858758	PL	82	F	20.1.20	2.2.20	Y	N	Y	N	Y	N	N	N	N	N				N	N	N	100/60	120	92	20	R	Y	N	N	N	Y	N	13	15	204	45		42	0.8	6.2	3.6	235	TROP-T +					Y	AF	MOD		Y			N	
124	882506	SS	65	M	17.01.20	22.01.20	Y	N	N	COUGH	N	Y	N	N	N	N				ILD	N	N	110/70	110	95	22	R	N	N	N	N	Y	N	13	9	208	60		48	1.14	6.7	3.21	400						Y			MOD		Y				
125	213857	AS	58	M	15.1.20	26.1.20	Y	Y	N	PEDAL EDEMA	Y	Y	N	N	N	N				N	N	N	130/70	102	96	18	N	N	N	N	N	Y	N	11	8	198	23		38	0.9	6.1	3	200						Y	Y		MOD					D	
126	801264	KK	55	M	18.1.20	28.1.20	Y	N	N	N	Y	Y	N	N	N	N	Y			N	N	N	140/80	89	95	20	N	N	Y	N	N	N	N	8	12	180	48		156	6	6.2	2.9	987									MILD						
127	30911	VB	35	F	15.1.20	17.1.20	N	Y	Y	N	N	N	N	N	N	N				N	N	N	120/70	101	97	18	N	N	Y	N	N	N	N	11	11	234	40	16	23	0.6	6.5	3.5	49		Y											N	
128	880462	MK	70	F	13.1.20	21.1.20	Y	N	N	N	N	N	Y	N	N	N				N	N	N	110/70	65	95	20	R	N	N	N	Y	Y	Y	12	12	532	68		59	0.8	6	2.76	501									SEVERE				Y	D	
129	785999	GK	44	F	13.1.20	25.1.20	Y	Y	Y	N	N	N	N	N	N	N				N	N	N	130/70	89	96	20	N	N	Y	N	N	N	Y	9	17	180	70		47	1.1	6	2.9	15									MILD					N	
130	550494	NA	49	F	8.1.20	16.1.20	Y	N	Y	N	N	N	N	N	SLE	Y				N	N	N	110/70	85	92	22	R	N	N	N	Y	Y	Y	9.4	5.3	136	80	46	200	8	5.6	2.8	1101							Y	SEVERE		Y		Y	D	Y
131	873629	S	57	F	4.2.20	11.2.20	Y	N	N	PEDAL EDEMA	Y	N	N	N	N	Y				N	N	N	140/80	102	89	20	R	N	Y	N	N	N	N	7.7	10	155	52		98	7.33	7	3.3	2000					Y				MILD					N	
132	877499	MK	36	F	5.2.20	10.2.20	Y	N	N	COUGH	N	N	N	N	N	N				RHD	N	N	120/70	110	95	19	R	N	N	N	N	Y	Y	9.7	5.2	152	47		58	1.2	7.3	3.09	566						Y			MOD					D	
133	877119	L	61	F	22.2.20	28.2.20	Y	N	N	PALPITATIONS	N	N	N	N	N	N	Y		Y	N	N	N	110/70	70	98	18	N	N	N	N	N	Y	N	4.2	15	162	59		103	2.81	6.5	3.9	890		Y				Y			MOD					N	
134	874206	H	32	M	15.2.20	24.2.20	N	N	Y	PAIN ABDOMEN	N	N	N	N	N	N				N	N	N	100/60	84	97	17	N	N	Y	N	N	N	N	7.1	1.6	99	78		79	2.17	5.6	2.5	180		Y							MILD						

SR NO	MRD NO	PERICARDIAL FLUID ANALYSIS (IF INDICATED)																		CECT/ MRI FINDINGS	BASIC DIAGNOSIS					MEDICAL TREATMENT									PERICARDIOCENTESIS	SURGICAL TREATMENT		COMMENT	COVID
		LVEF	LVDD TYPE	OTHER	TYPE (HEMORRHAGIC/ STRAW COLORED/ CHYLOUS/ OTHER)	SMEAR FOR AFB PLEURAL FLUID	MALIGNANT CYTOLOGY	CYTOLOGY			BIOCHEMISTRY				FLUID CULTURE	FLUID PH	CB-NAAT	LDH	OTHERS			MALIGNANCY	AUTOIMMUNE	OTHER	ASPIRIN	COLCHICINE	IBUPROFEN	CORTICOSTEROIDS	ATT	RRT	DIURETICS	OTHER		PLEURO-PERICARDIAL WINDOW	PERICARDIECTOMY		
								TLC	LYMPHOCYTES	NEUTROPHILS	GLUCOSE	PROTEIN	FLUID PRO/ S.PRO	ADA																							
91	1106186	30	III																Y	TUBERCULAR	PERICARDIAL EFFUSION				75					Y								
92	1110	55																	Y	TUBERCULAR	PERICARDIAL EFFUSION									Y							disseminated TB	
93	1109550	60																		HYPOTHYROIDISM	PERICARDIAL EFFUSION																	
94	67727	50	I																	PYOGENIC	PERICARDIAL EFFUSION																	ab+
95	1112169	60			STRAW	N	N	1000	75	25	1	5.2	0.87	139	N	7.5	N			TUBERCULAR	PERICARDIAL EFFUSION			RHD				WYSOLONE 30MG	Y				Y				ab+
96	1025978	42		IW & RVMI																post MI	ACUTE PERICARDITIS			IWMI	150						Y								
97	1028083	48	II	RWMA															Y	post MI	PERICARDIAL EFFUSION				150			WYSOLONE 30MG			Y							
98	64818	32																		HFrEF	PERICARDIAL EFFUSION			BIVENTRICULAR FAILURE							Y							
99	1054167	60																	Y	IDIOPATHIC	PERICARDIAL EFFUSION			PULMONARY TTHROMBOEMBOLISM								ENOXAPARIN						rtpcr+
100	1035436	30		RWMA																post MI	ACUTE PERICARDITIS				1950						Y								
101	517854	32	II	RWMA																post MI	ACUTE PERICARDITIS				75						Y								
102	1124199	45	III	GHK OF LV &RV																HFrEF	PERICARDIAL EFFUSION			BIVENTRICULAR FAILURE				EFFCORLIN			Y						DAMA	
103	1117128	38		RWMA																post MI	PERICARDIAL EFFUSION				75							TICAGRELOR	Y						
104	1123900	28		RWMA																post MI	PERICARDIAL EFFUSION				75						Y								
105	1121836	36	I																	IDIOPATHIC	ACUTE PERICARDITIS				75						Y								
106	1127677	32		RWMA																post MI	ACUTE PERICARDITIS				75						Y	TICAGRELOR							
107	1026573	38		RWMA																post MI	ACUTE PERICARDITIS				75						Y								
108	1000252	35		RWMA																post MI	ACUTE PERICARDITIS				150						Y								
109	998399	60																		HYPOTHYROIDISM	PERICARDIAL EFFUSION				75														
110	964910	28		RWMA																post MI	ACUTE PERICARDITIS				1950							CLOPIDOGREL							
111	1128213	22	II	GHK OF LV &RV																post MI	PERICARDIAL EFFUSION				150						Y							LOCALISED	
112	1124360	25	I	RWMA																HFrEF	PERICARDIAL EFFUSION				75						Y								
113	1115045	60		ANNULUS PARADOX															Y	IDIOPATHIC	CONSTRICTIVE PERICARDITIS																DAMA/ANNULUS PARADOXUS	
114	763166	20	III	DCMP																HFrEF	PERICARDIAL EFFUSION																	
115	634648	60	II																	HFpEF	PERICARDIAL EFFUSION				75	0.5 MG BD											GOUT	ab+
116	1121064	60			HEMORRHAGIC	N	ATYPICAL CELLS	80	20	80	21	5.3	0.82	307	N	7.2	N	5855		MALIGNANCY	PERICARDIAL EFFUSION	NHL						WYSOLONE				CHEMOTHERAPY	Y	Y		BIOPSY-NHL	
117	1131552	60			STRAW	N	N	2800	80	20	51	4.8	0.7	152	N	7.5	Y	764		TUBERCULAR	PERICARDIAL EFFUSION								Y				Y					
118	980469	60																		TUBERCULAR	ACUTE PERICARDITIS							WYSOLONE	Y									
119	1029222	50			STRAW	N	N	100	30	70	106	4.7	0.65	26	N	7.2	N	167		UREMIC	EFFUSIVE-CONSTRICTIVE							WYSOLONE			Y		Y				ab+
120	1128226	42			HEMORRHAGIC	N	N	2000	10	90	188	5.2	0.71	34	STAPH EPIDERMIDIS	7.3	N	617		PYOGENIC	PERICARDIAL EFFUSION										ANTIBIOTICS	Y					
121	1105479	60			STRAW	N	N	800	40	60	50	6.3	0.84	48	STAPH HEMOLYTICUS	7	N	205		PYOGENIC	PERICARDIAL EFFUSION										ANTIBIOTICS	Y					
122	1031343	60			HEMORRHAGIC	N	N	1000	5	95	44	5.7	0.97	28	N	7.5	N			MALIGNANCY	PERICARDIAL EFFUSION	CA BREAST									ANTIBIOTICS	Y					
123	858758	48		RWMA															Y	MALIGNANCY	PERICARDIAL EFFUSION	CA BREAST			150			WYSOLONE			Y	CHEMOTHERAPY						
124	882506	60		COR PULMONALE																HFpEF	PERICARDIAL EFFUSION							WYSOLONE			Y	DIGOXIN						
125	213857	38	III	SEVERE AS																HFrEF	PERICARDIAL EFFUSION				75						Y								
126	801264	60																		UREMIC	ACUTE PERICARDITIS									Y								
127	30911	60																		IDIOPATHIC	ACUTE PERICARDITIS				150			MEDROL										
128	880462	60			STRAW	N	N	500	90	10	38	5.1	0.85	152	N	7.5	N	1326		TUBERCULAR	PERICARDIAL EFFUSION				75			WYSOLONE	Y				Y					
129	785999	60																		MALIGNANCY	ACUTE PERICARDITIS	CA BREAST			75			WYSOLONE			Y							
130	550494	60			STRAW	N	N		30	70				20	N	7.4	N	577		AUTOIMMUNE	PERICARDIAL EFFUSION		SLE		75			WYSOLONE		Y	Y		Y					
131	873629	60																		UREMIC	ACUTE PERICARDITIS				75					Y								
132	877499	45																		HFrEF	PERICARDIAL EFFUSION				75						Y								
133	877119	60																		HYPOTHYROIDISM	PERICARDIAL EFFUSION				75			MEDROL										
134	874206	60																		MALIGNANCY	ACUTE PERICARDITIS	GERM CELL TUMOUR						WYSOLONE										

							CHIEF COMPLAINTS				MEDICAL CO-MORBIDITIES										H/O CARDIAC SURGERY	PREVIOUS H/O PERICARDIAL DISEASE	CLINICAL EXAMINATION						PERICARDIAL RUB	PERICARDIAL KNOCK	PULSUS PARADOXUS	CHEST X-RAY		LAB FINDINGS												ECG FINDINGS						2D ECHO FINDINGS						
SR NO	MRD NO	NAME	AGE	SEX	DOA	DOD	SOB	CHEST PAIN	FEVER	OTHER	DM	HTN	IHD	TB	MALIGNANCY	AUTOIMMUNE	CKD	COPD	HYPOTHYROIDISM	OTHER			BP	HR	SP02	RR	JVP	LAD				CARDIOMEGALY	PLEURAL EFFUSION	HB	TLC	PLATELETS	ESR	CRP	B.UREA	CREATININE	TOTAL PROTEINS	ALBUMIN	BNP	OTHERS	ST SEGMENT CHANGES	PR SEGMENT CHANGES	ELECTRICAL ALTERNANS	T WAVE LOW/ FLATTENED/ INVERTED	LOW VOLTAGE ECG	OTHERS	MILD/ MODERATE/ SEVERE PERICARDIAL EFFUSION	THICKENED PERICARDIUM	EXUDATIVE STRANDS/FRONDS	EFFUSIVE-CONSTRICTIVE	CARDIAC TAMPONADE	IVC COLLAPSE/ NORMAL/ DISTENDED	RESPIRATORY VARIATION OF MV & TV
135	1154047	PR	65	F	16.6.21	23.6.21	Y	N	N	N	Y	Y	N	N	N	N				N	N	N	120/70	99	86	20	R	N	N	N	Y	Y	9.3	10	373	100		34	0.9	5.5	2.99	79						Y		SEVERE					D	
136	1156383	KK	60	F	17.6.21	21.6.21	Y	Y	N	N	Y	Y	N	N	N	N				N	N	N	140/90	112	93	22	R	N	N	N	Y	N	10	13	185			93	1.9	7.7	4.3								SEVERE				Y		
137	949415	S	65	M	31.7.20	5.8.20	Y	N	N	↓UO	Y	N	N	N	N	N				N	N	N	100/60	97	87	22	R	N	N	N	Y	Y	10	11	201	35		34	1.2	6	3.5	780		Y					MILD					D	
138	690440	AR	73	F	9.3.20	14.3.20	Y	N	N	VOMITING	N	N	Y	N	N	N			Y	N	N	N	140/90	80	95	19	N	N	N	N	N	Y	7.6	7.6	276	50		46	1.6	6.9	3.9	970						LBBB	MOD					N	
139	802428	SK	66	F	14.7.20	24.7.20	N	N	N	PALPITATIONS	Y	Y	Y	N	N	N				N	N	N	110/70	138	89	20	R	N	N	N	N	Y	13	9	155	20		59	1	7.1	4.1	903						AF	MOD					D	
140	940914	SD	55	F	16.8.20	5.9.20	N	N	N	GBW	Y	Y	N	N	N	N	Y		Y	N	N	N	80/50	56	70	18	N	N	N	N	N	N	6.7	8.5	128	67		25	2.12	6.7	3.5	1200					Y		MILD					N	
141	938385	AK	63	F	28.6.20	6.7.20	Y	Y	N	N	Y	Y	N	N	N	N				N	N	N	80/60	154	85	21	N	N	N	N	N	N	12	13	206	52		63	1.5	6.8	3.66	730		Y				RWMA	MILD						
142	587600	MS	50	M	17.2.20	25.2.20	Y	N	N	PEDAL EDEMA	Y	Y	N	N	N	N	Y			N	N	N	150/90	120	94	18	N	N	Y	N	N	N	7	13	179	35		67	8	6.1	3.1	900						Y	MILD					N	
143	899719	IS	55	M	15.2.20	20.2.20	N	Y	N	N	Y	Y	N	N	N	N				N	N	N	130/80	87	95	19	N	N	N	N	N	N	14	12	302	10		23	0.8	7.3	4.2			Y				RWMA	MILD						
144	507118	JS	49	M	11.2.20	25.2.20	N	N	Y	PAIN ABDOMEN	N	N	N	N	N	N				N	N	N	100/60	105	97	16	N	N	N	N	N	N	9.7	9.9	497	59		30	1	7	4	80		Y					MILD						
145	889614	VR	44	F	10.2.20	15.2.20	N	Y	N	N	N	N	N	N	N	N				N	N	N	110/70	89	97	17	N	N	Y	N	N	N	9.7	6.2	253	33		15	0.9	7.1	4.5	67							MILD						
146	150046	AR	57	F	9.2.20	16.2.20	Y	N	N	N	Y	N	N	N	N	N				N	N	N	120/70	110	96	22	R	N	N	N	N	N	10	8	301	20		59	1.7	6	3	600							MILD					D	
147	893695	IP	60	M	8.2.20	14.2.20	Y	N	N	COUGH	N	N	N	N	N	N				N	N	N	90/60	116	88	18	N	N	N	N	N	Y	9.9	7.9	179	34		43	0.7	6.2	2.95	1230		Y			Y		MOD					D	
148	922491	M	35	M	15.4.20	20.4.20	Y	N	N	PEDAL EDEMA	N	N	N	N	N	N				RHD	N	N	130/80	129	97	22	R	N	N	N	N	Y	10	11	231	23		40	1.1	6.9	3.8	589						AF	MILD						
149	920464	P	18	M	31.3.20	3.4.20	Y	N	Y	COUGH	N	N	N	N	N	N				THALASSEMIA	N	N	100/60	110	96	18	R	N	N	N	N	Y	8	12	330	48		30	0.8	6	3	34						Y	SEVERE		Y		Y	D	Y
150	920342	PS	62	M	6.6.20	12.6.20	N	N	N	ALTERED SENSORIUM	Y	N	N	Y	N	N				N	N	N	110/60	86	95	17	N	N	N	N	N	Y	9	18	201	67		45	1	5.6	2.8	120		Y			Y	Y	MOD						
151	879476	PK	48	F	2.6.20	13.6.20	N	N	Y	PAIN ABDOMEN	N	N	N	N	N	N				N	N	N	120/70	113	94	19	N	N	N	N	N	Y	9	29	110	45	78	43	1.1	6	3.4	70						Y	MOD						
152	876509	BK	77	F	26.4.21	13.5.21	Y	Y	N	N	N	Y	Y	N	N	N	Y	Y		N	N	N	160/80	118	90	18	N	N	N	N	N	Y	10	13	394		69	83	1.7	6.1	3.5							AF	MILD						
153	21371	P	36	F	23.5.21	27.5.21	Y	N	Y	N	N	N	N	N	N	N				THALASSEMIA	N	N	100/60	98	95	19	N	N	Y	N	N	N	8	13	206	39		28	0.9	5.6	2.9			Y											
154	818489	J	46	F	17.4.21	23.4.21	Y	N	N	GBW	N	Y	N	N	N	N	Y			N	N	N	140/80	104	97	16	N	N	N	N	N	N	8	8	328	14	8	100	4	5.9	2.4	450							MOD		Y	Y			Y
155	1095014	SK	60	M	27.4.21	30.4.21	N	N	Y	GBW	Y	N	N	N	N	N				N	N	N	120/70	78	96	16	N	N	N	N	N	N	12	9	276	80		42	1.2	6.7	4	13							MILD		Y				
156	1048364	PS	64	F	19.4.21	27.4.21	Y	N	Y	ABDOMINAL DISTENSTION	N	N	N	N	N	N				N	N	N	110/70	90	94	19	N	N	Y	N	N	N	7.8	15	187	67		39	1	6	3.2	300		Y	Y				MILD					D	
157	501029	KS	59	M	13.4.21	22.4.21	N	N	N	↓UO	Y	Y	Y	N	N	N	Y			N	N	N	80/50	123	80	22	N	N	Y	N	N	N	9	20	178	70		87	6	5.6	3	800							MILD						
158	1085351	AR	29	F	14.4.21	21.4.21	N	N	Y	COUGH	N	N	N	N	N	SLE	Y			N	N	N	100/60	122	94	17	N	N	N	N	N	Y	7.7	5	122	70		28	0.67	4.7	1.5							Y	MOD						Y
159	1078641	SS	71	M	9.4.21	13.4.21	Y	N	Y	N	Y	N	Y	N	N	N				N	N	N	130/80	78	96	18	N	N	N	N	N	N	11	3.8	80	38		25	0.8	6.6	2.8	78						Y	MILD					C	
160	1079578	MD	77	F	8.4.21	15.4.21	Y	N	N	N	N	Y	N	N	N	N			Y	N	N	N	120/70	80	94	16	N	N	N	N	N	N	10	8	178	38		30	0.9	6.7	4	149						Y	MILD					D	
161	568862	VK	72	M	9.4.21	15.4.21	Y	N	Y	GBW	Y	Y	N	N	N	N	Y			N	N	N	150/90	101	95	20	N	N	N	N	N	Y	7.7	7.4	116	67		129	5.49	5.4	2.7	880					Y		MILD						
162	1095833	VK	55	M	25.4.21	9.5.21	Y	N	Y	COUGH	N	Y	N	N	N	N	Y			N	N	N	130/80	121	89	19	R	N	N	N	N	N	9	12	60	44	34	46	3	6	3	200					Y		MILD						
163	1110600	RS	49	M	16.6.21	22.6.21	Y	N	N	N	N	N	N	N	N	N				SEVERE AS	N	N	100/60	98	95	17	N	N	N	N	N	N	13	7.3	187	6		63	1.2	5.6	3.1	5000		Y					MILD					D	
164	1111821	KL	53	F	21.5.21	27.5.21	Y	N	N	N	N	N	N	N	N	N				N	N	N	130/70	94	96	22	N	N	N	N	N	Y	12	8.9	140	38		20	0.5	6	3.1						Y	Y	MOD				Y		
165	1101090	PK	43	F	24.5.21	3.6.21	Y	N	N	N	Y	Y	Y	N	N	N				N	N	N	100/60	110	95	19	N	N	Y	N	N	N	11	11	352	110	26	20	0.4	6.7	3.42	584							MILD						
166	1043006	SS	64	F	14.3.21	29.3.21	Y	N	N	N	N	N	N	N	N	N				N	N	N	110/70	89	96	20	N	N	N	N	N	N	14	23	467	20		47	0.53	6.3	3.7								MILD						
167	1046424	MS	50	M	9.3.21	3.4.21	Y	N	Y	GBW	Y	N	N	N	N	N				N	N	N	90/60	84	92	21	R	N	N	N	N	Y	11	7.5	255	45		95	1.8	6.7	3.84	676						LBBB	MILD					D	
168	1039804	SK	45	F	4.3.21	8.3.21	N	Y	N	N	N	Y	N	N	N	N	Y			N	N	N	120/70	88	96	16	N	N	Y	N	N	N	9	8.2	243			106	6.7	6.6	3.5								MILD						
169	1209764	PK	54	M	18.6.21	24.6.21	Y	N	N	↓UO	Y	Y	N	N	N	N	Y			N	N	N	140/90	90	95	19	R	N	N	N	N	Y	4.3	18	175			122	6.75	6	2.9	900					Y		SEVERE				Y		
170	1048800	JK	73	M	12.3.21	17.3.21	Y	Y	N	N	N	Y	N	N	N	N	Y			SEVERE AS	N	N	130/70	102	94	20	R	N	N	N	N	N	7.2	3.8	70			57	4.15	5.6	2.9			Y			Y		MILD						

SR NO	MRD NO				PERICARDIAL FLUID ANALYSIS (IF INDICATED)															CECT/ MRI FINDINGS	BASIC DIAGNOSIS					MEDICAL TREATMENT									SURGICAL TREATMENT				
		LVEF	LVDD TYPE	OTHER	TYPE (HEMORRHAGIC/ STRAW COLORED/ CHYLOUS/ OTHER)	SMEAR FOR AFB PLEURAL FLUID	MALIGNANT CYTOLOGY	CYTOLOGY			BIOCHEMISTRY				FLUID CULTURE	FLUID PH	CB-NAAT	LDH	OTHERS			MALIGNANCY	AUTOIMMUNE	OTHER	ASPIRIN	COLCHICINE	IBUPROFEN	CORTICOSTEROIDS	ATT	RRT	DIURETICS	OTHER	PERICARDIOCENTESIS	PLEURO-PERICARDIAL WINDOW	PERICARDIECTOMY	COMMENT	COVID
								TLC	LYMPHOCYTES	NEUTROPHILS	GLUCOSE	PROTEIN	FLUID PRO/ S.PRO	ADA																						
135	1154047	60			STRAW	N	N	1000	70	30	159	5.5	1	51	N	7.4	N	879			TUBERCULAR	PERICARDIAL EFFUSION							WYSOLONE	Y				Y				
136	1156383	60			HEMORRHAGIC	N	ATYPICAL CELLS	2000	80	20	173	3.8	0.49	42	N	7.3	N	1435		Y	MALIGNANCY	PERICARDIAL EFFUSION	BRONCHOGENIC CA						EFFCORLIN					Y				
137	949415	14	III																		IDIOPATHIC	PERICARDIAL EFFUSION				75						Y						
138	690440	28	III																		HYPOTHYROIDISM	PERICARDIAL EFFUSION				75						Y						
139	802428	25																			HFrEF	PERICARDIAL EFFUSION				150						Y						
140	940914	45	II																		UREMIC	PERICARDIAL EFFUSION				75						Y						
141	938385	35	III																		post MI	ACUTE PERICARDITIS				150						Y						
142	587600	45																			UREMIC	ACUTE PERICARDITIS				75					Y	Y						
143	899719	30	I																		IDIOPATHIC	ACUTE PERICARDITIS				150			WYSOLONE									
144	507118	60																			IDIOPATHIC	ACUTE PERICARDITIS				150			WYSOLONE									
145	889614	60																			IDIOPATHIC	ACUTE PERICARDITIS							OMNACORTIL									
146	150046	58	I																		HFpEF	PERICARDIAL EFFUSION				75						Y						
147	893695	28		RWMA																	HFrEF	PERICARDIAL EFFUSION				150						Y						
148	922491	30																			HFrEF	PERICARDIAL EFFUSION				75						Y						
149	920464	60			STRAW	N	N	1000	80	20	6	3.6	0.6	62	N	N	N	780			TUBERCULAR	PERICARDIAL EFFUSION							WYSOLONE	Y				Y				
150	920342	28		RWMA/LOCALISED																	TUBERCULAR	PERICARDIAL EFFUSION				75			EFFCORLIN	Y		Y					TBM/DAMA	
151	879476	60																			MALIGNANCY	PERICARDIAL EFFUSION	GIST						WYSOLONE			Y						
152	876509	60																			IDIOPATHIC	ACUTE PERICARDITIS							MEDROL			Y						ab+
153	21371	60																			IDIOPATHIC	ACUTE PERICARDITIS					0.5											
154	818489	50																			UREMIC	EFFUSIVE-CONSTRICTIVE				75					Y	Y						
155	1095014	60		LOCALISED																	TUBERCULAR	PERICARDIAL EFFUSION							WYSOLONE	Y							MANTOUX +	
156	1048364	42	II	RWMA																	IDIOPATHIC	ACUTE PERICARDITIS				75			MEDROL			Y					EXPIRED	
157	501029	34																			UREMIC	ACUTE PERICARDITIS				150					Y	Y					DAMA	
158	1085351	60																			AUTOIMMUNE	PERICARDIAL EFFUSION		SLE					WYSOLONE				MMF,HCQ					
159	1078641	36																			OTHER	PERICARDIAL EFFUSION			DENGUE													
160	1079578	60	II																		HYPOTHYROIDISM	PERICARDIAL EFFUSION																
161	568862	60																			TUBERCULAR	PERICARDIAL EFFUSION							WYSOLONE	Y							PLEURAL TB	
162	1095833	45		GHK OF LV																	OTHER	PERICARDIAL EFFUSION			DENGUE				EFFCORLIN			Y						
163	1110600	18																			HFrEF	PERICARDIAL EFFUSION				75						Y						ab+
164	1111821	60			STRAW	N	N	100	75	25	95	5.5	0.92	62	N	7.4	N	564		Y	TUBERCULAR	PERICARDIAL EFFUSION							WYSOLONE	Y				Y			PULMONARY KOCH'S	
165	1101090	35		RWMA																	IDIOPATHIC	ACUTE PERICARDITIS				75			WYSOLONE			Y						rtpcr+
166	1043006	60																			MALIGNANCY	PERICARDIAL EFFUSION	CA BREAST						WYSOLONE				CHEMOTHERAPY					
167	1046424	20		DCMP																	HFrEF	PERICARDIAL EFFUSION				75						Y						
168	1039804	45		GHK OF LV															LOCALISED		UREMIC	ACUTE PERICARDITIS									Y	Y						
169	1209764	45			STRAW	N	N	1000	30	70	109	5.3	0.88	27	N	7.5	N	719			UREMIC	PERICARDIAL EFFUSION									Y	Y		Y				
170	1048800	35		RWMA																	UREMIC	ACUTE PERICARDITIS			150						Y							

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Date 20/4/2021

The Principal,
DMC,Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Dr. Karanjot Singh**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical profile of pericardial diseases

Supervisor
Dr. Sandeep Chhabra
Assoc. Prof.,
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Co-Supervisor
Dr. Narender Pal Jain
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The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

CLINICAL PROFILE OF PERICARDIAL DISEASES

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

IN

DEPARTMENT OF MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

NOVEMBER, 2019

DR. KARANJOT SINGH

DEPARTMENT OF MEDICINE
DAYANAND MEDICAL COLLEGE & HOSPITAL
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ABSTRACT OF PLAN OF THESIS

Title	CLINICAL PROFILE OF PERICARDIAL DISEASES
For the degree of	MD MEDICINE
Name of the candidate	Dr. Karanjot Singh
Supervisor	Dr. Sandeep Chhabra, Associate Professor, Department of Medicine, DMCH, Ludhiana
Co-supervisor	Dr. Narender Pal Jain, Professor, Department of Medicine, DMCH, Ludhiana Dr. Shibba Takkar, Associate Professor, Department of Cardiology, DMCH, Ludhiana.
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: This is observational cross-sectional study in all consecutive patients admitted in DMC&H and HDHI with findings suggestive of any pericardial disease or with history of pericardial disease or with incidental finding of pericardial disease.

Aim of Study:

1) To study the clinical spectrum of pericardial diseases

2) To study etiopathogenesis, clinical and echocardiographic profile of patients with pericardial disease.

Materials & Methods: This is an observational cross-sectional study and duration of study will be from January 2020 to June 2021.

Clinical Significance: To identify the etiopathogenic factors, echocardiographic and clinical profile of patients with pericardial disease.

Keywords: Pericarditis, Tamponade, Pericardiocentesis, Constrictive, Effusion.

(Signature) Candidate	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis entitled "**CLINICAL PROFILE OF PERICARDIAL DISEASES**" exist in the Department of Medicine, Dayanand Medical College & Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

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Date : _____

APPROVAL PROFORMA
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DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Karanjot Singh
Department	Medicine
Topic of Thesis	Clinical profile of Pericardial diseases
Likely date of appearing in PG examination	May-June 2022
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Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Karanjot Singh PG Resident in Department of Medicine, DMC & H , Ludhiana
2	NAME OF THE INSTITUTE	Dayanand Medical College and Hospital
3	COURSE OF STUDY AND SUBJECT	Doctor of Medicine in Department of Medicine
4	DATE OF ADMISSION OF COURSE	16-05-2019
5	TITLE OF THE TOPIC	Clinical Profile of Pericardial Diseases

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Pericardial disease is a common disorder seen in varying clinical settings and may be the first manifestation of an underlying systemic disease and can be due to multiple causes. New diagnostic techniques have improved diagnosis, allowing early diagnosis and management but there are few randomized data to guide physicians in the management of pericardial diseases.[1]

Deciding on the extend of diagnostic evaluation requires good clinical judgement based on a careful evaluation of the risk-benefit ratio, and knowledge of basic epidemiological data for management plan. Epidemiological studies are lacking, and the exact incidence and prevalence are unknown.[2]

In clinical practice a probabilistic approach seems reasonable: in Western countries most cases are idiopathic or viral with a brief and benign course. On contrary, in developing countries like India specific pericarditis such as tuberculous pericarditis is quite common and should be ruled out.[2]

Systemic inflammatory diseases represent a large group of rare diseases that may involve all organs and also the heart. The three layers of heart can be affected. Modern sophisticated imaging techniques reveal cardiac abnormalities in most of these diseases and the cardiac involvement ranges widely depending upon the applied diagnostic methods and selection of patients. The clinical significance of these findings is not always established. Better knowledge and awareness of cardiac involvement is necessary[3].

Relapsing or recurrent pericarditis is probably the most troublesome complication of acute pericarditis, and represents one of the greatest therapeutic challenges among the disorders of the pericardium. The etiology and pathogenesis of idiopathic recurrent acute pericarditis (IRAP) remain controversial standing like a bridge that crosses infectious, autoimmune and auto-inflammatory pathways.[4]

Chronic pericardial effusions are a major cause of morbidity in some clinical settings. Although the treatment of choice for acute symptomatic pericardial effusions (tamponade) is pericardiocentesis, the long term management of symptomatic chronic pericardial effusions provide a greater challenge.[5]

Neoplastic pericardial effusion is a serious and common clinical disorder encountered by cardiologists, cardiothoracic surgeons, oncologists, and radiation oncologists. It may develop from direct extension or metastatic spread of the underlying malignancy, from an opportunistic infection, or from a complication of radiation therapy or chemotherapeutic toxicity and the clinical presentation varies.[6]

Pericardial effusion is a common finding in everyday clinical practice. The first challenge to the clinician is to try to establish an etiologic diagnosis. Sometimes, the pericardial effusion can be easily related to a known underlying disease, such as acute myocardial infarction, cardiac surgery, end-stage renal disease or widespread metastatic neoplasm and sometimes no obvious cause is apparent.[7]

Pericardial involvement in end-stage renal disease (ESRD) is manifested most commonly as acute uremic or dialysis pericarditis and infrequently as chronic constrictive pericarditis. The causes of uremic and dialysis pericarditis remain uncertain.[8]

Several common misconceptions can make the clinical diagnosis of subacute pericardial tamponade challenging. Widely known physical findings of pericardial tamponade lack sensitivity and specificity. Interpretation of echocardiographic signs requires good understanding of pathophysiology. Over-reliance on echocardiography may result in over-utilization of pericardial drainage procedures. Awareness of these misconceptions with an integrative approach to both clinical and imaging data will help clinicians to assess the hemodynamic impact of pericardial effusion and the need for drainage.[9]

6.2 REVIEW OF LITERATURE

Wang Z F et al concluded that infection, neoplasm, injury, and congenital disease all may affect the pericardium. Although echocardiography is the primary imaging modality used to examine the pericardium, CT and MRimaging provide various advantages, especially in cases of loculated or hemorrhagic effusion, constrictive pericarditis, and pericardial masses. CT and MR imaging should be used when findings at echocardiography are difficult to interpret or conflict with clinical findings..[10]

Zayas R et al done study to access the specific etiology and the role of methods for specific etiologic diagnosis in patients with primary acute pericarditis. Study done on 100 patients with primary acute pericarditis, a general protocol was performed in all patients, whereas only pericardiocentesis was performed in patients with clinical cardiac tamponade or an unfavourable course with anti-inflammatory drugs. Surgical drainage and pericardial biopsy was performed in patients with tamponade relapse. A specific etiology was discovered in 22 patients (22%) (neoplasm in 7, tuberculosis in 4, other infections in 3, collagen diseases in 3, thyroid disorder in 4 and dissecting aortic aneurysm in 1). The general diagnostic protocol led to specific diagnosis in 15 patients (68% of all patients with specific acute pericarditis) and pericardiocentesis in other 7 patients (32%). The role of a diagnostic protocol, therapeutic pericardiocentesis, and diagnostic pericardiocentesis was similar and complementary. Pericardial biopsy results were negative in the 5 patients in whom it was performed. Cardiac tamponade and an unfavourable clinical outcome were significant($p<0.001$) associated with the finding of a specific etiology; when both features were combined, sensitivity was 86% and specificity 85 %, positive predictive value 63% and negative predictive value 96%. Conclusion was that the specific etiology in patients with primary acute pericarditis is about 20% to 25%, and that about 90% of these specific cases can be discovered by using the described systemic diagnostic protocol only in patients with an unfavourable outcome(cardiac tamponade or poor clinical outcome).[11]

Pankuweit S et al concluded that bacterial pericarditis occurs by direct infection during trauma, thoracic surgery, or catheter drainage, by spread from an intrathoracic, myocardial, or subdiaphragmatic focus, and by hematogenous dissemination. The frequent causes are Staphylococcus and Streptococcus (rheumatic pancarditis), Haemophilus, and M. tuberculosis. In AIDS pericarditis, the incidence of bacterial infection is much higher than in the general population, with a high proportion of Mycobacterium avium-intracellulare infection. Purulent pericarditis is the most serious manifestation of bacterial pericarditis, characterized by gross pus in the pericardium or microscopically purulent effusion. It is an acute, fulminant illness with fever in virtually all patients. Chest pain is uncommon. Purulent pericarditis is always fatal if untreated. The mortality rate in treated patients is 40%, and death is mostly due to cardiac tamponade, systemic toxicity, cardiac decompensation, and constriction. Tuberculous infection may present as acute pericarditis, cardiac tamponade, silent (often large) relapsing pericardial effusion, effusive constrictive pericarditis, toxic symptoms with persistent fever, and acute, subacute, or chronic constriction. The mortality in untreated patients approaches 85%.[12]

Brucato A et al showed that Idiopathic recurrent acute pericarditis (IRAP) occurs in up to 20–50% of patients and represents the most troublesome complication of acute pericarditis. It is generally idiopathic or post-cardiac injury. IRAP is a disease of suspected immune-mediated pathogenesis. On the other hand, it has been suggested that some of these patients might have an atypical or subclinical form of an auto-inflammatory disease, e.g. genetic disorders characterized by primary dysfunction of the innate immune system and caused by mutations of genes involved in the inflammatory response. IRAP often needs a multidrug therapy: NSAIDs or aspirin at high dosages every 6–8 h, corticosteroids only rarely, at low dosages and with a very gradual tapering (months) and colchicine at low dosages if tolerated. Anakinra could be a solution for patients who do not tolerate other therapies.[4]

Miralda G P showed that in patients with acute pericarditis and features suggesting associated disease the likelihood of this being the cause for the pericardial syndrome is very high. Therefore, associated disease should be investigated accordingly. In the immunologically competent patient presenting with classically acute pericarditis, the probability of acute idiopathic pericarditis is about 90%, this probability being even greater when clinical features subside in the following few days. No further studies are needed in such cases. In a study of our group, tamponade was significantly more common in patients with tuberculous or purulent pericarditis or neoplasia, but the high prevalence of acute idiopathic pericarditis made it the leading cause in absolute terms. However, in tamponade without findings of inflammation (chest pain or rub) we have demonstrated a likelihood ratio of 3.0 for neoplasia, although other etiologies should be considered as well. Other features such as a sustained clinical course (for example, over three weeks) also increase the likelihood of specific disease. However, in our part of the world, the predominance of idiopathic disease in any presentation of acute pericarditis and no associated conditions may be taken as a clinical rule. An exception is tamponade with massively haemorrhagic effusion, often but not always caused by malignancy.In addition, purulent pericarditis should always be considered in predisposing diseases (such as pleural empyema or mediastinal infection), even in the absence of definite features of pericarditis or tamponade.[13]

Wander G S, Chabbra S T concluded that the most frequent causes of relapsing pericarditis are idiopathic or viral pericarditis and post pericardial injury syndromes. In patients without previous cardiac surgery, and in whom connective tissue disease has been ruled out, relapsing-intermittent pericarditis with symptom free periods longer than six weeks makes the diagnosis of idiopathic/viral pericarditis practically certain. The clinical evolution is characterised by the recurrence of the episodes of acute pericarditis, which frequently are progressively less severe, without clinical tamponade, and without evolution to constrictive pericarditis. Fortunately, only few cases develop the incessant type of relapsing pericarditis. The treatment

modalities include anti-inflammatory therapy - Corticosteroids, NSAIDS, Aspirin, Colchicine & immunosuppressives (Refractory cases). Interventional techniques like pericardiectomy, pericardial window an intra pericardial administration of triamcinolone have limited role.[14]

Bonnefoy E, Godon P et al concluded in patients with idiopathic acute pericarditis, an increase in cardiac troponin I is frequently observed, especially in younger patients and those with a recent infection. Although ST-segment elevation does not reliably indicate myocardial injury, a significant cardiac troponin-I increase is only seen in these patients.[15]

Imazio M et al concluded that specific clinical features (fever -38°C, subacute course, large effusion or tamponade, and aspirin or NSAID failure) may be useful to identify higher risk of specific causal conditions and complications in acute pericarditis[16].

Spodick DH concluded that acute pericarditis has a wide spectrum of etiologies. Modern advances have reduced the number of infectious cases and restricted the ECG stage sequence. The NSAIDs, especially ibuprofen, recently with the addition of colchicine, have been rapidly effective in most appropriately diagnosed cases. However, misinterpretation as other disease, especially acute myocardial infarction, pulmonary embolism, and heart failure with tamponade is common. Doppler echocardiographic recording has largely solved the differential diagnosis of pericardial effusion and aided the recognition of cardiac tamponade[17].

Rehman KA et al concluded that with improvement in dialysis techniques

and mandatory monitoring of solute clearance, earlier initiation of hemodialysis, and efficient identification of advanced CKD patients, the incidence of uremic pericarditis and dialysis pericarditis has been reduced. Hence, when uremic pericarditis is diagnosed, the dialysis regimen for the patient should be revisited. Optimization of water and electrolyte imbalance, acid–base homeostasis through dialysis,remains

the mainstay of treatment and prevention of this subset of pericarditis[18].

Meyers D G et al showed that in pericardial effusion exudates differed from transudates by higher leukocyte counts and ratios of fluid to serum lactate dehydrogenase levels. Fluid glucose levels were significantly less in exudates. Sensitivity for detecting exudates was high for specific gravity > 1.015 (90%), fluid total protein>3.0 g/dL (97%), fluid to serum protein ratio >0.5 (96%), fluid lactate dehydrogenase ratio >0.6(94%), and fluid to serum glucose ratio < 1.0 (85%). None of these indicators were specific. Fluid total protein and specific gravity were moderately correlated (r=0.56). Fluid cytologic study had a sensitivity of 92% and specificity of 100% for malignant effusion. No other test was diagnostic for a specific etiology. Among infection-associated effusions, culture-positive fluid had more neutrophils, higher lactate dehydrogenase levels, and lower ratios of fluid to serum glucose than culture-negative (parainfective)fluid[19].

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical spectrum of pericardial diseases.
- To study etiopathogenesis, clinical and echocardiographic profile of patients with pericardial disease.

7.MATERIALS AND METHODS

7.1 Source of Data

All consecutive patients admitted in DMC&H and HDHI with findings suggestive of any pericardial disease or with history of pericardial disease or with incidental finding of pericardial disease.

7.2 METHOD OF COLLECTION OF DATA

All the IPD patients fulfilling the inclusion criteria will be included in this study. The designed proforma will be used to record the specific details including history, clinical findings, lab investigations, ecg findings, 2D echo findings, pericardial fluid analysis if indicated, radiological findings, basic diagnosis and management of the cases of pericardial disease during this period. The treatment modalities will be according to the discretion of treating physician.

INCLUSION CRITERIA

- All consecutive patients age above 18 years with findings suggestive of pericardial diseases (acute pericarditis, pericardial effusion, cardiac tamponade, chronic pericarditis, constrictive pericarditis, recurrent pericarditis, etc) will be included.

EXCLUSION CRITERIA

- All patients less than 18 years.
- Post CABG and post ICR (intra-cardiac repair) patients with post-operative effusions in primary hospital admission.

DURATION OF STUDY: Duration of study will be from January 2020 to June 2021

STATISTICAL ANALYSIS: Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

No

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached along with

8. LIST OF REFERENCES

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PROFORMA

SR NO. MRD NO.

NAME AGE SEX

ADDRESS

PHONE NO

DOA DOD

Chief complaints

Medical co-morbidities(DM/HTN/IHD/TB/Malignancy/Autoimmune/Other)

H/o cardiac surgery

Previous h/o pericardial disease

Clinical examination:

BP HR spO2 RR JVP LAD

Pericardial rub

Pericardial knock

Pulsusparadoxus

Chest X Ray findings(PA/AP/Lateral)

Signature of Candidate **Signature of supervisor**

Lab findings:

- Hemoglobin
- TLC
- P/L/M/E/B
- Platelets
- ESR
- CRP
- BUN
- S. creatinine
- Total proteins
- Albumin
- Others

ECG findings

- ST segment changes
- PR segment changes
- Electrical alternans
- T wave low/flattened/inverted
- Low voltage ecg
- Others

Signature of Candidate **Signature of supervisor**

2D echo finding

- Mild/Moderate/Severe Pericardial effusion
- Thickened pericardium
- Exudative strands
- Effusive-constrictive
- Cardiac tamponade
- IVC collapse/normal
- Respiratory variation of MV & TV velocities
- Other

Pericardial fluid analysis (if indicated)

- Type(Hemorrhagic/straw colored/chylous/other)
- Smear for AFB Pleural fluid
- Malignant cytology
- Fluid biochemistry
- ADA
- Fluid culture
- Fluid PH
- CB-Naat
- Others

CECT/MRI findings

Signature of Candidate **Signature of supervisor**

Ultrasound abdomen (if indicated)

Basic Diagnosis

Management

- Medical treatment

Drug	Dosage	Route	Duration
1. Aspirin			
2. Colchicine			
3. Ibuprofen			
4. Corticosteroids			
5. Other			

- Pericardiocentesis

  Duration of pigtail(if indicated)

  Amount of fluid aspirated

- Surgical treatment

Comment :

Signature of Candidate **Signature of supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....

am willing to participate in the study **"CLINICAL PROFILE OF PERICARDIAL DISEASES"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried cut. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"CLINICAL PROFILE OF PERICARDIAL DISEASES"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"CLINICAL PROFILE OF PERICARDIAL DISEASES" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ